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Original Articles

FAMILIAL FACTORS IN DIAGNOSIS, TREATMENT, AND PROGNOSIS OF DENTOFACIAL DISTURBANCES*

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THE PROBLEM

THE problems of this paper are: (1) to ascertain what features of the dentition and supporting structures are transmitted by the genetic mechanism; (2) to determine the *modus operandi* of these genetic attributes in the production of dentofacial disturbances; and (3) to evaluate their role in diagnosis, treatment, and prognosis of these disturbances. It is impossible, for the moment at least, to conduct experimental genetics on humans in the same sense we do on animals. However, human beings mate extensively and intensively enough that conditions equivalent to experimental animal genetics are obtained, provided controlled techniques for their study can be established. These controls are mainly empirical and statistical. This fact makes the problem of data collection and analysis difficult, but, even so, it does not invalidate the proposition that human genetics can be studied and that conclusions can be drawn.

Although nongenetic factors are important in the formation of facial structures, these are beyond the consideration of this paper. It is an error to presume that genetic factors can ever exist independent of environment or to insist that environmental influences cannot affect or even seriously modify a

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structure of genetic origin. The intentional omission of a discussion of nurtural factors which also may be involved does not imply they are regarded as unimportant nor does it imply that subsequent work should omit a serious consideration of their interactive roles.

A review of our early data revealed to us that we were guilty of the common error of regarding certain broad classes of malocclusion as entities. For example, we were inclined to accept the occurrence of such anomalies as Class II malocclusion in parents and offspring as being significant, even suggestive of the operation of genetic factors. It was not long, however, until we noted that in families possessing Class II malocclusion, these Class II malocclusions might each exhibit a different set of physical attributes. One might be the result of a combination of normal maxilla, normal mandibular body, and short mandibular rami; another might be the result of deficient maxilla, deficient mandibular body, and short mandibular rami; still another might be the result of normal maxilla, normal mandibular rami, and short mandibular body. It began to appear reasonable that malocclusion or normal occlusion, as such, need not be directly inherited since evidence appeared to support the belief that the dentofacial complex is made up of numerous genetic attributes. It became apparent that if we were to be able to demonstrate how dentofacial anomalies fit into the genetic picture, it would be necessary for us to observe in detail the morphologic traits of the entire dentofacial complex.

For this reason, about four years ago we refined our method of approach to the problem of testing heredity's role in malocclusion by formalizing, systematizing, and particularizing the collection of data.

THE LITERATURE

Papers published in dental periodicals within the last fifteen years furnish adequate evidence that the genetics of the dentofacial structures is regarded as increasingly important in etiology, diagnosis, and treatment of dental disturbances. However, a stock-taking of the literature reveals the major obstacle to an understanding of the importance of hereditary traits to be a lack of adequate data upon which to formulate hypotheses and to draw conclusions. Following are representative comments. Many others could be cited following the same line of thought. Guyer[1] in 1924 summarizes the status of available evidence:

> After trying to get together the available facts regarding the part played by heredity in dental and facial abnormalities, I find myself in much the frame of mind of the quarrelsome negro described in one of the late Ambassador Page's letters. This negro tried in vain to exasperate a fellow negro into a fight. In spite of the storming and swearing of the first negro the other remained stubbornly silent. Finally negro Number 1 burst out with, "Look here, you kinky-headed, flat-nosed, slab-footed nigger, I warns you 'fore God, don't you keep givin' me none o' your damned silence."
>
> Like the vexed negro, I have found silence—or in other words the absence of reliable data—a great source of irritation. Opinions in this field, I find in abundance, but clear-cut unequivocal evidence, backed up by specific cases, is scarce. I have no doubt that some of the opinion in question is correct; nevertheless, it cannot be accepted as scientific truth until the facts upon which it is based are brought forward and subjected to the searchlight of modern genetics.

In 1927 Phillips[2] presents the same argument:

> Of heredity, and by it I refer to the transmutation of type only, we know so little, that I will not attempt to say anything, other than to point to certain striking characteristics of the enamel of the teeth, in which heredity, I think, plays an important part.

The same opinion is iterated by Brash[3] in 1929:

> There comes indeed a point in history of any subject, in which much of the discussion has been of a speculative nature, when it is essential to clear the ground; to examine critically general conclusions which are currently repeated; to probe the basis of facile hypothesis; and face with frankness the sometimes not very welcome fact that speculation, though it may on occasion anticipate discovery, is no substitute for inquiry.
>
> The reading of many papers and a sifting out of opinions and counter-opinions have been, I may be allowed to remark, very stimulating to the critical faculty, and have left a very clear impression on my mind that very little positive evidence has ever been adduced in support of any of the many supposed causes of irregularity and malocclusion. I have indeed found so little of positive knowledge and so much speculation that it has been clear to me, ever since I began to contemplate a discussion of the subject, that it would be necessary to review every aspect of it on fundamental principles and with strict reference to evidence.
>
> It is equally clear, so far as satisfactory conclusions on the inheritance of irregularities and malocclusions of the teeth are concerned, that the necessary data are simply not available.

Charles[4] in 1931 said:

> The relative positions of heredity and environment in growth are still the subject of considerable difference of opinion, principally, I think, because so very little is known about them.

Johnson (1933)[5] indicates evidence of heredity in occlusion to be available, enabling one to formulate some broad generalizations. These factors are of rather indeterminate specificity, however. Crouch (1935)[6] believes, "Heredity doubtless plays an important role in predetermining malocclusions or tendencies toward such malocclusions," but does not state what these hereditary factors are.

Casto[7] (1935) believes:

> The effect of heredity in producing malformation of the jaws and malocclusions of the teeth has been a controversial question for many years. There is, moreover, no present indication that the question will ever be definitely settled.

The textbook *Practical Orthodontia* by Dewey (revised in 1935 by Anderson) fails to reveal specific knowledge of the heredity of traits in the dentofacial complex to be known. As a matter of fact it lists as congenital causes some traits which are believed to be hereditary by other authors.

Goldstein and Stanton[8] (1936) are not specific and say:

> The factor of heredity, however, is undoubtedly the direct cause of malocclusion in specific instances, and, as Brash points out, adequate investigation in this field is lacking, and is much needed.

The same year Sly[9] writes:

> There is probably no more widely discussed or more controversial question than that which concerns the role of heredity in the causation of malocclusion; and while the present knowledge of the subject is insufficient to permit of its final solution, nevertheless some expression of opinion regarding it is essential to any consideration of the biologic factor in orthodontia.

The report of Johnson[10] on the results of cross breeding contrasted types of dogs at Cornell Experimental Farm provides us with the type of well-documented information vital to the understanding of the role of genetics in occlusion and necessary to the formulation of operative procedures which recognize the importance of etiology in the outlining of treatment policies for individual cases. Although this work was done on dogs Johnson's statement is to the point and merits consideration.

> It is true that in skull types and dental occlusion the human being is quite different from the dog. The application of the results of animal experimentation to human requires bridging a gap which to some orthodontists raises grave doubts. Be this as it may, the principles of genetics, so far as we know them today, apply to all living organisms. The nature of chromosomal distribution, the appearance of dominants and recessives, and the problem of mutant factors have been studied in plants and animals of various kinds, and they reveal the same basic phenomena. Man is no exception. Moreover, until proof to the contrary can be adduced, it is safe to assume that the biologic relationship of the teeth to their supporting structures is much the same in man as in the dog.

A review of the literature dealing with the inheritance of traits of the dentofacial complex and of the role of inheritance in etiology, diagnosis, and treatment of dental disturbances leaves the authors in substantial agreement with the writers previously quoted. Many of the articles dealing with the role of heredity in occlusion have been based upon empirical and logical argument and have been inadequately documented in their presentation of supporting evidence. It has been assumed that forces which operate in the biologic world are equally operative in the human world, and, the supporting evidence from experimental genetics is adequate to warrant the belief that genetic factors are both extensive and of basic importance in any form having the property of life. There is no question at this point of the operation of heredity in man. That such genetic factors operate, and extensively, is now established with reasonable scientific probability. Consequently, the problem of further research is to isolate specific attributes, to determine their mode of transmission, to acquire knowledge concerning their development, and to understand how they are affected by the environment in which they must exist. To attack this problem successfully, the first requisite is adequate data.

Although a review of the literature reveals data inadequacy with reference to the broader problems of occlusion, there are many individual attributes in the dentofacial complex whose genetics is moderately well known. For the most part these traits are special characters pertaining to the teeth themselves

and tend to be anomalous, such as absence of certain teeth, absence of certain groupings of teeth, absence of all teeth of one denomination, absence or deficient roots of teeth, diminutive maxillary incisors, noneruption of maxillary canines, hypoplasia of enamel, microdontia, incisor teeth erupted at birth, supernumerary teeth, susceptibility to dental caries, and so on. There are over a hundred references in the literature to the heredity of characters such as these and, on the whole, the details of transmission are fairly well understood. For the most part, however, the characters mentioned above are conspicuous, aberrant, and poorly represented in the population at large. In addition, although these traits are important in the etiology, diagnosis, and treatment of malocclusions which they engender or of which they may be a part, they do not make up the bulk of malocclusions and certainly the majority of treatment cases do not involve factors which can be attributed to these anomalous traits. As a matter of fact, a review of the literature would warrant an expression of the opinion that unless a character is conspicuous and unusual it is likely to be regarded as nonhereditary. In other words, there seems to be a feeling that the normal is immune to genetic laws; this obviously is absurd.

To date, most of the genetic work in orthodontics has been done by studying family pedigrees or by detailed examination of identical twins, comparing them with siblings. The former method lends itself admirably to the study of the conspicuous and anomalous traits; this largely because one can assume that the information presented by the untrained observer is satisfactorily accurate. Probably inaccuracy of observation rather than lack of family pedigree material has made conclusions on the more general problems of occlusion of doubtful validity. People have observed and have commented upon the fact that children resemble parents or earlier progenitors with reference to the face as a whole and there has been a belief that if the parents were normal or the familial line "untainted" with reference to almost any attribute the children would be more likely to be normal than if abnormalities or "taints" appeared within a family. These observations undoubtedly have foundation in fact, and implicit in the conclusions drawn from them is the assumption that genetic factors are operative. However, the majority of conclusions drawn from this type of unformulated observation have been extremely varied and, for the most part, erroneous. Consequently, they have had but little practical value to the orthodontic profession.

Weinberger[11] (1926) lists some of these generalizations:

> Perhaps the most common of generalizations obtained from data of the above-mentioned kinds is the belief that a child inherits his traits or features from some specific member of the family. For example, there has been a wide-spread belief that (1) children resemble their mothers more than they do their fathers and that the mother due to the fact that she nurtures a child during the fetal period, thus contributes much more strongly to the heredity of the child than does the father; (2) that the males show predominant resemblance to the mothers while the father contributes the majority of hereditary items to the females; (3) children resemble grandparents more than parents

> —here again is the belief that the maternal grandparent contributes more strongly to the heredity of the male grandchildren, while the paternal grandparent contributes more strongly to the heredity of the female grandchildren; (4) that heredity is most clearly shown in resemblance between children and parental siblings. In other words, heredity is presumed to operate in family lines with different progenitors or ancestors occupying positions of primary importance. Most of these ideas have largely disappeared from the field at the present time; however, there is one current belief that still has a number of followers, that is the belief that mothers contribute more strongly to the inheritance of children than do fathers.
>
> Another wide-spread belief apparently obtaining from this same type of uniformulized and noncritically tested evidence has been the hypothesis that hereditary malocclusions are more difficult, if not impossible, to treat. As early as 1864 Cartwright states that of two types of underhung jaws one condition is fortuitous and is amenable to mechanical treatment and the other condition is hereditary or congenital and cannot be remedied.

Furthermore, the continuance of controversy regarding the relative importance of heredity or environment in the production of malocclusion can be attributed to the continued reliance of professional men upon this type of evidence and upon their willingness to maintain argument without critically examining the relevancy of fact to opinion. Since this point of view has been extant for well over fifty years in orthodontics and since it has seriously hindered the advancement of understanding, we must again emphasize the primary problem to be a careful collection of material upon which to exercise analytical and critical acumen before presenting conclusions.

METHOD

For many years we have been recording in the orthodontic clinic at the University of Michigan as much data concerning parents and siblings of our patients as we could conveniently obtain, but never with much system. Our observations were concerned mainly with special characters such as those previously mentioned, and our notes on dentofacial anomalies were gross in respect to the anomaly as a whole rather than minute in respect to those component parts whose individual characteristics made harmonious occlusal relation impossible. An evaluation of these unsystematized data clearly demonstrated to us the need for a more detailed examination of the face and its component parts and emphasized the importance of systematic observation and recording if our materials were to be of value for analysis and comparison.

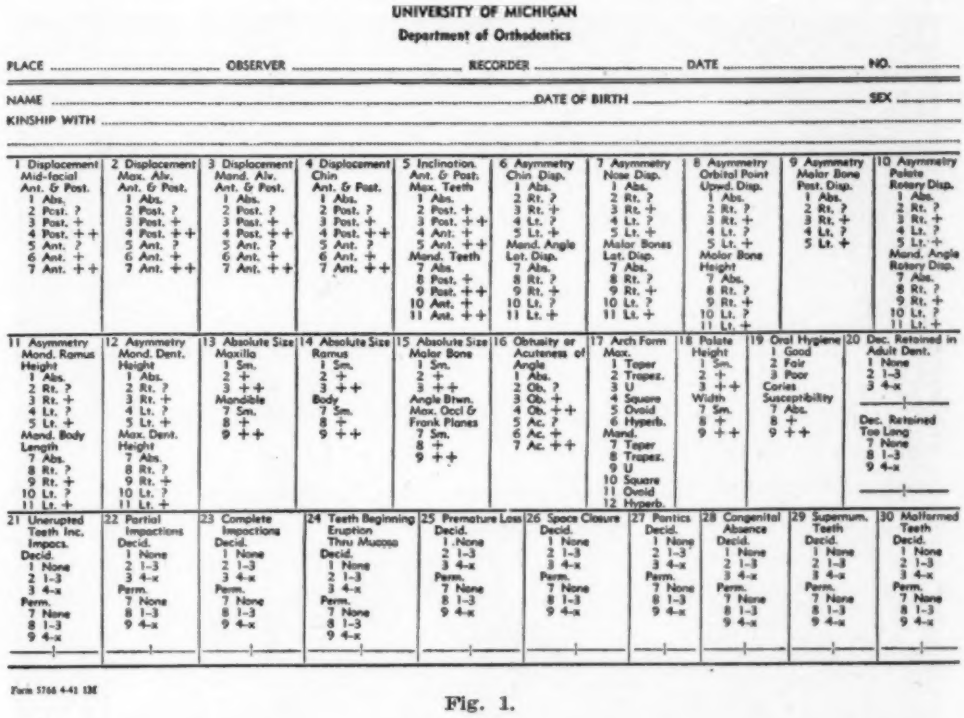
Many of the facial features, which seemed to us important, were not amenable to measurement. These were observed and recorded in a graded series.* Hooton[12] defends this method, "Morphological features which can be observed and described but cannot be measured are probably of greater anthropological significance than diameters and indices."

*The evaluation of detailed attributes of the dentofacial complex must be the work of orthodontists since clinical significance of dentofacial features can be properly appreciated only by men who have made a special study of them. Because orthodontists, as a rule, lack expert knowledge of genetic methodology and biometrical analysis, precise and effective presentation can best be secured by collaboration.

Examination Technique

After many months of preliminary record taking, the examination sheet shown in Figs. 1 and 2 was evolved.

Heading numbers one to four on our examination form refer to excesses or deficiencies in anteroposterior dimensions of the midfacial, maxillary alveolar, mandibular alveolar, and chin regions, respectively (Figs. 3 and 4). All four are taken from a profile view and at first it is surprising to note how



UNIVERSITY OF MICHIGAN
Department of Orthodontics

PLACE ..... OBSERVER ..... RECORDER ..... DATE ..... NO. .....

NAME ..... DATE OF BIRTH ..... SEX .....

KINSHIP WITH .....

1 Displacement Mid-facial Ant. & Post.	2 Displacement Max. Alv. Ant. & Post.	3 Displacement Mand. Alv. Ant. & Post.	4 Displacement Chin Ant. & Post.	5 Inclination Ant. & Post. Max. Teeth	6 Asymmetry Chin Disp.	7 Asymmetry Nose Disp.	8 Asymmetry Orbital Point Upwd. Disp.	9 Asymmetry Molar Bone Post. Disp.	10 Asymmetry Palate Rotary Disp.
1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.
2 Post. ?	2 Post. ?	2 Post. ?	2 Post. ?	2 Post. +	2 Rt. ?	2 Rt. ?	2 Rt. ?	2 Rt. ?	2 Rt. ?
3 Post. +	3 Post. +	3 Post. +	3 Post. +	3 Post. ++	3 Rt. +	3 Rt. +	3 Rt. +	3 Rt. +	3 Rt. +
4 Post. ++	4 Post. ++	4 Post. ++	4 Post. ++	4 Ant. +	4 Lt. ?	4 Lt. ?	4 Lt. ?	4 Lt. ?	4 Lt. ?
5 Ant. ?	5 Ant. ?	5 Ant. ?	5 Ant. ?	5 Ant. ++	5 Lt. +	5 Lt. +	5 Lt. +	5 Lt. +	5 Lt. +
6 Ant. +	6 Ant. +	6 Ant. +	6 Ant. +	Mand. Teeth	Mand. Angle	Molar Bones	Molar Bone		Mand. Angle
7 Ant. ++	7 Ant. ++	7 Ant. ++	7 Ant. ++	7 Abs.	Lat. Disp.	Lat. Disp.	Height		Rotary Disp.
				8 Post. +	7 Abs.	7 Abs.	7 Abs.		7 Abs.
				9 Post. ++	8 Rt. ?	8 Rt. ?	8 Rt. ?		8 Rt. ?
				10 Ant. +	9 Rt. +	9 Rt. +	9 Rt. +		9 Rt. +
				11 Ant. ++	10 Lt. ?	10 Lt. ?	10 Lt. ?		10 Lt. ?
					11 Lt. +	11 Lt. +	11 Lt. +		11 Lt. +

11 Asymmetry Mand. Ramus Height	12 Asymmetry Mand. Dent. Height	13 Absolute Size Maxilla	14 Absolute Size Ramus	15 Absolute Size Molar Bone	16 Obtusity or Acuteness of Angle	17 Arch Form Max.	18 Palate Height	19 Oral Hygiene	20 Dec. Retained in Adult Dent.
1 Abs.	1 Abs.	1 Sm.	1 Sm.	1 Sm.	1 Abs.	1 Taper	1 Sm.	1 Good	1 None
2 Rt. ?	2 Rt. ?	2 +	2 +	2 +	2 Ob. ?	2 Trapez.	2 +	2 Fair	2 1-3
3 Rt. +	3 Rt. +	3 ++	3 ++	3 ++	3 Ob. +	3 U	3 ++	3 Poor	3 4-x
4 Lt. ?	4 Lt. ?	Mandible	Body	Angle Btwn.	4 Ob. ++	4 Square	Width	Caries	
5 Lt. +	5 Lt. +	7 Sm.	7 Sm.	Max. Occl &	5 Ac. ?	5 Ovoid	7 Sm.	Susceptibility	Dec. Retained
Mand. Body	Max. Dent.	8 +	8 +	Frank Planes	6 Ac. +	6 Hyperb.	8 +	7 Abs.	Too Long
Length	Height	9 ++	9 ++	7 Sm.	7 Ac. ++	Mand.	9 ++	8 +	7 None
7 Abs.	7 Abs.			8 +		7 Taper		9 ++	8 1-3
8 Rt. ?	8 Rt. ?			9 ++		8 Trapez.			9 4-x
9 Rt. +	9 Rt. +					9 U			
10 Lt. ?	10 Lt. ?					10 Square			
11 Lt. +	11 Lt. +					11 Ovoid			
						12 Hyperb.			

21 Unerupted Teeth Inc. Impacs.	22 Partial Impactions	23 Complete Impactions	24 Teeth Beginning Eruption Thru Mucosa	25 Premature Loss	26 Space Closure	27 Pontics	28 Congenital Absence	29 Supernum. Teeth	30 Malformed Teeth
Decid.	Decid.	Decid.	Decid.	Decid.	Decid.	Decid.	Decid.	Decid.	Decid.
1 None	1 None	1 None	1 None	1 None	1 None	1 None	1 None	1 None	1 None
2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3	2 1-3
3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x	3 4-x
Perm.	Perm.	Perm.	Perm.	Perm.	Perm.	Perm.	Perm.	Perm.	Perm.
7 None	7 None	7 None	7 None	7 None	7 None	7 None	7 None	7 None	7 None
8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3	8 1-3
9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x	9 4-x

Form 5766 4-41 12M

Fig. 1.

31 Hypoplasia of Enamel	32 Crowding & Spacing	33 Crowding & Spacing	34 Rotated Teeth (++ only)	35 Depth of bite	36 Anteriors in X-bite	37 Rt. Buccal Teeth in X-bite	38 Lt. Buccal Teeth in X-bite	39 Buccal Quadrant Completely Ling. to Opponent
Decid.	Up. Ants.	Up. Buc. Teeth	Decid.	Decid.	Decid.	Decid.	Decid.	1 Abs.
1 None	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	1 Abs.	2 Rt. max.
2 1-3	2 Cr. +	2 Cr. +	2 Pres.	2 Deep +	2 Pres.	2 Pres.	2 Pres.	3 Lt. max.
3 4-x	3 Cr. ++	3 Cr. ++	Perm.	3 Deep ++	Perm.	Perm.	Perm.	4 Rt. mand.
Perm.	4 Sp. +	4 Sp. +	7 Abs.	4 Deep +++	7 Abs.	7 Abs.	7 Abs.	5 Lt. mand.
7 None	5 Sp. ++	5 Sp. ++	8 Pres.	5 Open +	8 Pres.	8 Pres.	8 Pres.	Completely Buccal
8 1-3	Lo. Ants.	Lo. Buc. Teeth		6 Open ++				7 Abs.
9 4-x	7 Abs.	7 Abs.		Perm.				8 Rt. max.
	8 Cr. +	8 Cr. +		7 Abs.				9 Lt. max.
	9 Cr. ++	9 Cr. ++		8 Deep +				10 Rt. mand.
	10 Sp. +	10 Sp. +		9 Deep ++				11 Lt. mand.
	11 Sp. ++	11 Sp. ++		10 Deep +++				
				11 Open +				
				12 Open ++				

40 Occlusion	41 Class I (Angle)	42 Class II (Angle)	43 Class III (Angle)	44 Oral Habits
1 Normal	1 Abs.	1 Abs.	1 Abs.	1 Abs.
2 Border Line	2 Growth case	2 Div. 1 Bilat. (Typ.)	2 Bilateral	2 Th. Suck.
3 Abnormal	3 Non-growth case	3 Div. 1 Bilat. (Atyp.)	3 Unilateral	3 Fi. Suck.
	4 Max. mesial drift	4 Div. 1 Unilateral	4 Mutilated	4 Tg. Suck.
	5 Mand. mesial drift	5 Div. 2 Bilat. (Typ.)		5 Ck. Suck.
	6 Mutilated	6 Div. 2 Bilat. (Atyp.)		6 Ck. Bite
	7	7 Div. 2 Unilateral		7 U. lp. Bite
	(other)	8 Mutilated		8 L. lp. Bite
				9 Nail Bite
				10 Nite Gr.
				11 Mouth Br.
				12 .....
				(other)

45 Disposition of Case	46 Disposition of Case	47 Treatment Indicated	49 Records Available	50	51
1 No attention indicated	1 Assign to Grad. for study	1 Comprehensive	1 Operative	1	1
2 Observation indicated	2 Assign to Grad. for obs.	2 Compromise	2 Anthroponometric	2	2
3 Treatment indicated	3 Assign to Grad. for treatment	3 Palliative	3 Radiographic, complete	3	3
4 Not admitted here	4 Assign to Grad. for	4 Preventive	4 Radiographic, partial	4	4
5 Place on waiting list with encouragement	(other)	5	5 Photographic, oriented facial	5	5
6 Place on waiting list without encouragement	5 Referred to	(other)	6 Photographic, unoriented facial	6	6
7 Assign to Soph. for observation	(own dentist)	48 Case Closed	7 Photographic, intraoral	7	7
8 Assign to Jr. for observation	6 Referred to		8	8	8
9 Assign to Jr. for treatment	(own orthodontist)		(other)	9	9
10 Assign to Sr. for observation	7 Referred to			10	10
11 Assign to Sr. for treatment	.....			11	11
12	.....			12	12
(other)	.....				
	(other dept's in D. S.)				

Remarks:

Fig. 2.

independent of each other these four levels of the face appear to be. Mid-face could be termed maxillary apical base and is meant to include maxillary (except alveolar processes), nasal, and zygomatic regions.

Column number five refers to inclination of the maxillary and mandibular anterior teeth regardless of their anteroposterior positions in the skull, and again judgment is based on knowledge of the average condition at any stage of development.

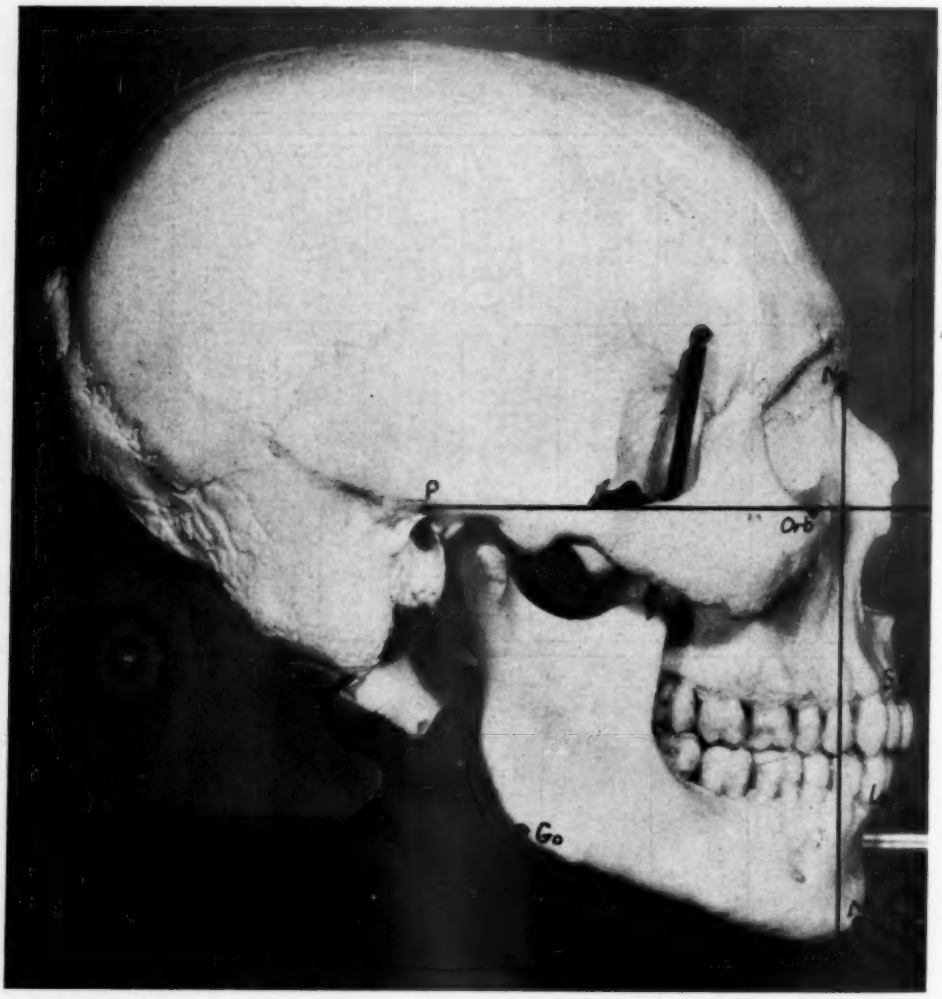


Fig. 3.

Figs. 3 and 4.—*Na.*, Nasion. *Orb.*, Orbital point. *P.*, Porion. *SP.*, Superior Prosthion. *IP.*, Inferior Prosthion. *Go.*, Gonion. The plane formed by the orbital and porion points is the Frankfort horizontal plane. The plane dropped from nasion perpendicular to the Frankfort plane is used for evaluating anterior and posterior displacements. Note in the figure that the maxillary apical base, the maxillary alveolus, the maxillary and mandibular anterior teeth, the mandibular alveolus and the mandibular body are all displaced considerably forward of the plane dropped from Nasion.

Thirteen categories on the record sheet refer to facial asymmetries. Attention has been called to these asymmetries; they have been measured often in the past by anthropologists using dry specimens. It is almost as easy to detect them on living as on dead material. Columns six to twelve, inclusive, record the thirteen different facial asymmetries (Figs. 5 and 6). Four are *lateral*

displacements: chin, mandibular angle, nose, and malar bone. Two are rotary displacements: palate and mandibular angle. Five refer to greater dimensions on the one side than on the other: malar bone, mandibular ramus, maxillary dental height, mandibular dental height, and mandibular body length. One records a higher level on one side: orbital point upward displacement. Finally, one records a more posterior relation of one side: malar bone posterior displacement.

Lateral displacements, orbital point upward displacement, and the various height differences are detected from a front view of the patient's face as he sits in the dental chair.

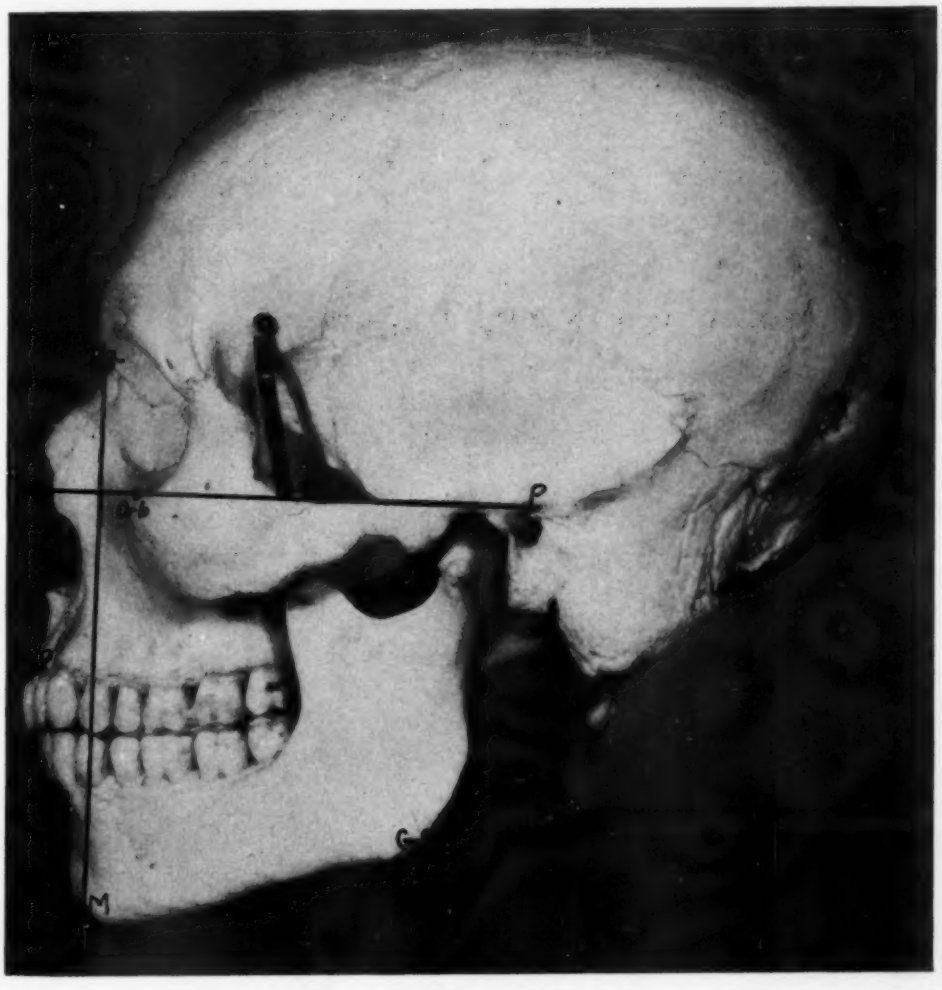


Fig. 4.

Usually palpation is necessary in order to evaluate asymmetry in height of malar bones and mandibular rami. Maxillary and mandibular dental height asymmetries are also detected by palpation. The points taken are occlusal plane to orbits for maxillary dental height and occlusal plane to inferior border of mandible for mandibular dental height.

Asymmetries of malar bone posterior displacement, mandibular angle rotary displacement, and mandibular body length are seen best by tipping the chair

back and viewing the head from below. Rotary asymmetry of the palate is present if a plane passing vertically through the posterior half of the raphe forms an angle other than 90 degrees with the orbital plane. This can be determined only by viewing the palate from directly below with the patient's mouth widely opened.

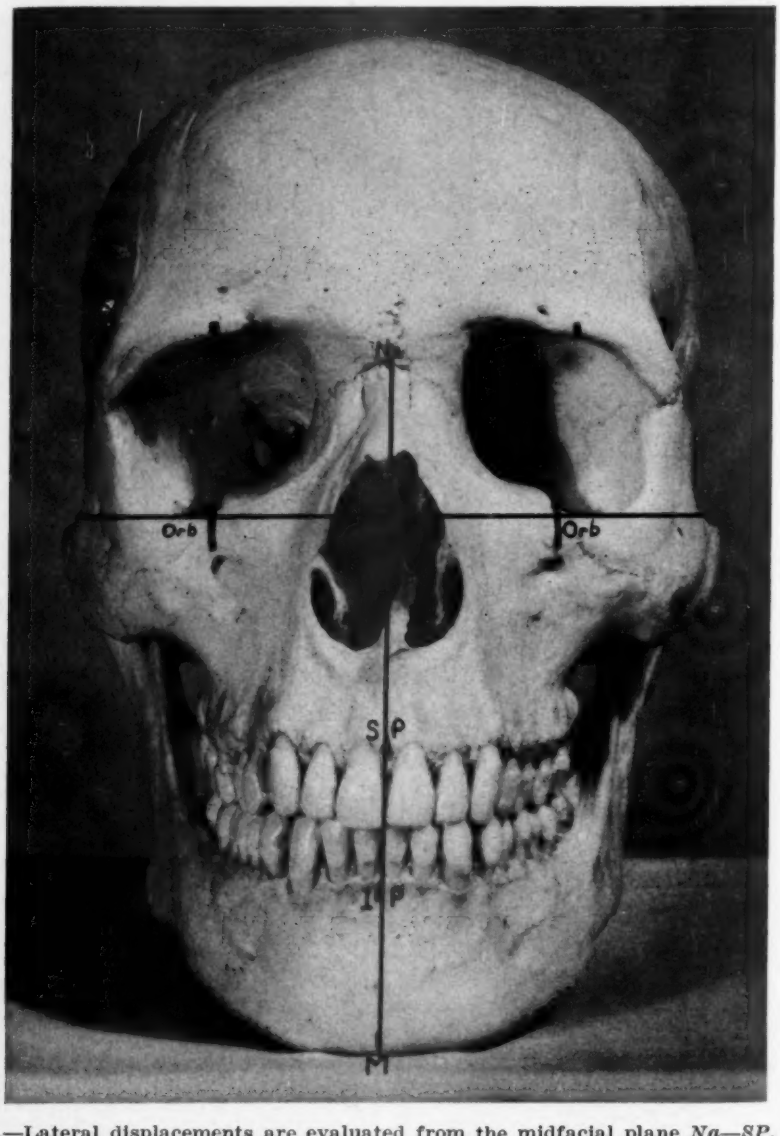


Fig. 5.—Lateral displacements are evaluated from the midfacial plane *Na—SP*. Very few lateral displacements are noticeable in this skull. Note that the plane *Na—SP* extends downward to include the midpoints of the mandible, *IP*. and *M*. Vertical asymmetries are evaluated from the orbital plane (*Orb—Orb*) and from the Frankfort horizontal plane. Here, again, few asymmetries are evident.

Headings 13, 14, 15, 16, 18, and 35 refer to absolute size of maxilla, mandible, rami, body, and malar bone; to angle between maxillary occlusal plane and Frankfort horizontal plane; to degree of obtusity or acuity of mandibular angle; to palate height; to palate width; and to depth of bite. Here, average for developmental stage of the individual under examination must form the basis of comparison in each of these.

Categories such as arch form, deciduous teeth retained, impactions, congenital absence, supernumerary teeth, hypoplasia of enamel, crowding, spacing, types of occlusion and malocclusion, and oral habits appear under twenty-six different headings. Observations in this entire group are absolute in that they are purely descriptive of the condition and are not dependent upon developmental stage.

The balance of the card is arranged for clinical and clerical convenience in our particular institution.

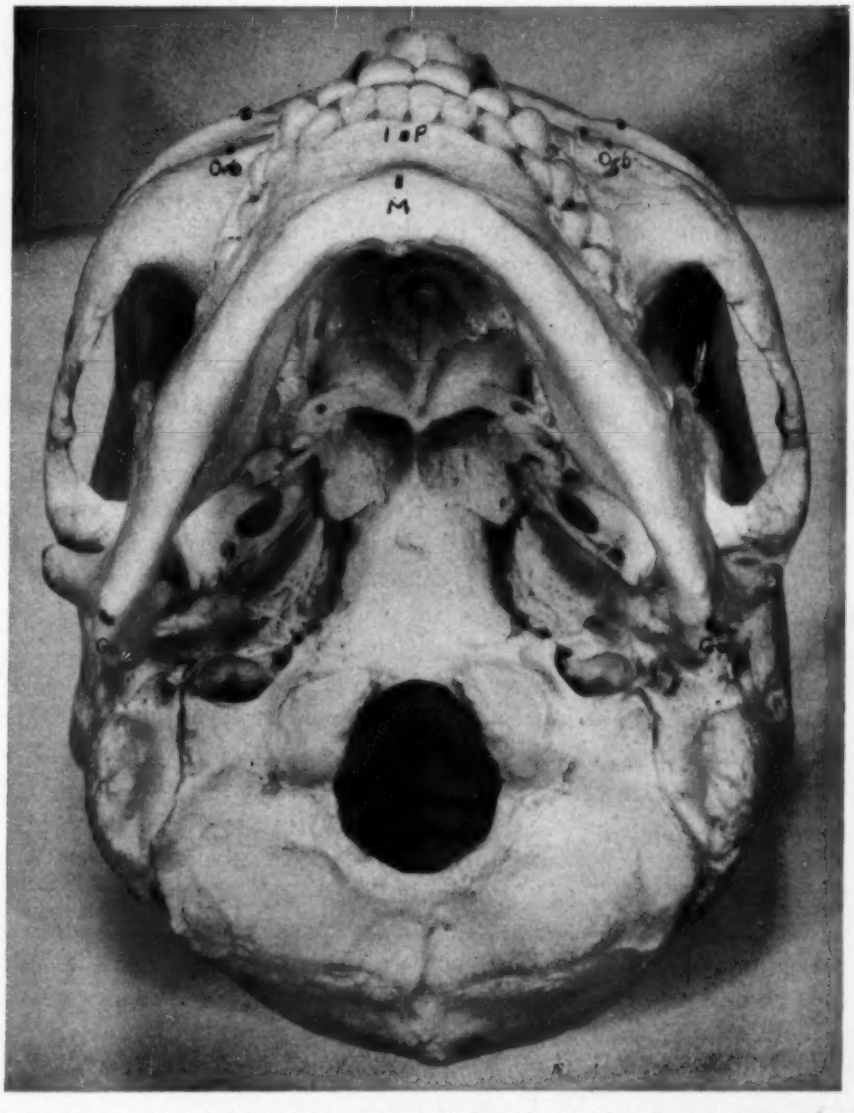


Fig. 6.—The patient is placed in approximately this position for appraising rotary and lateral displacements of the mandible. In this skull the mandible appears to be rotated slightly to the right. The degree of rotation is slight and probably not enough to be detected on the living.

THE SAMPLE

The data for this study include 265 individuals grouped in 78 families; 130 are children and 135 are parents. Dental and anthropometric records were secured on each individual. In addition supporting x-ray, photographic, and

clinical operative records were obtained on those individuals who were admitted to the clinic for treatment.

The population sample is selected and is not presumed to be representative of the population as a whole in the community from which the clinic draws its patients. At least one child in each family was brought to the orthodontic clinic because the parents were concerned over a real or apparent dentofacial disorder. Although this selection fails to give us much evidence of total population, it does give the very important professional advantage of being representative of a group that seeks orthodontic advice and treatment. This is the group which primarily concerns us in problems of etiology, diagnosis, and treatment.

AGE AND SEX

The male parents number 57, present an average age of 41.9 years, and an age range of 31 to 61 years; 73.3 per cent are between 35 and 45 years old; consequently the male parents may be classified as a middle-aged group. Female parents number 78, have an average age of 38.9 years and an age range of 29 to 55 years. Among females age is more variable than among males: 61.4 per cent are between 35 and 45 years, 24.3 per cent are under 35, and 14.3 per cent are over 45 years of age.

It is reasonable to assume that the longer environmental forces operate the more likely are nonhereditary factors to be interposed in the dentition and supporting structures. Some of these nonhereditary factors and their consequent effects are relatively easy to observe, others are exceedingly detailed and complicated. In all cases we have been careful to record the extent to which, in our opinion, these modifying traits have contributed to the dentofacial disturbance.

Three males (35, 43, 61 yr.) and 6 females (43, 44, 44, 47, 49, 55 yr.) present full upper and lower dentures and 3 females (37, 42, 44 yr.) have complete upper and partial lower dentures. One of these latter appeared to have been Class II: no classification of occlusion was made in the other cases.

Five males (8.8 per cent) and 8 females (10.3 per cent) are classified as normal. Class I malocclusions are shown in 36 males (63.2 per cent) and 42 females (53.8 per cent); mutilation of one kind or another has contributed to 22 of the male and to 29 of the female Class I's. The higher incidence of mutilation among the female Class I's appears to be significant. Males present 10 (17.5 per cent) Class II malocclusions and mutilation is a contributing factor in 5 of the cases. Females show 17 (21.8 per cent) Class II malocclusions with mutilation contributing to 6 of the cases. Class III malocclusions are limited in both sexes, 2 males and 2 females; mutilation is involved in 1 male case.

Among the parents, with the exception of those presenting full or partial dentures, the average age for the nonmutilated group is 2.7 years less than for those who show mutilation. This difference while small is significant in view of the fact that the majority of parents fall between 33 and 45 years of age.

The problem of time is as important among the children as it is among parents. Here the reason is different. From the point of view of development heredity becomes a part of a process that extends at least from conception to maturity. Most, if not all, hereditary features undergo vast change between

inception and maturity and it frequently is very difficult to describe the end product in terms of its appearance during the process of development. Time and space do not warrant further elaboration of this thesis. We want to emphasize, however, that the developmental process and heredity are integral.

In order to obtain an overview of some of the changes associated with the developmental process we have arranged the children in four groups. A further refinement will eventually be desirable but for the moment the following arrangement, although arbitrary, is convenient:

Group I. A full complement of deciduous teeth present with no permanent teeth erupted through the mucosa.

Group II. Begins with the eruption through the mucosa of any of the following teeth (a) $\frac{621\ /126}{621/\ 126}$ and terminates with the eruption through the mucosa of any of these teeth (b) $\frac{7543\ /3457}{7543/\ 3457}$.

Group III. Begins with (b) above and extends to eruption through mucosa of $\frac{8\ /8}{8/\ 8}$.

Group IV. Complete young adult dentition fully erupted.

The chronologic age in months sorted on the dentition stages is recorded in Table I. The age ranges included in each dentition stage are, on the whole, satisfactory for present purposes. Females show a somewhat greater developmental precocity than do males. This is a usual sex difference.

TABLE I

DENTITION STAGES AND CHRONOLOGIC AGE IN MONTHS

DENTITION STAGE	MALE			FEMALE		
	NO.	RANGE	MEAN	NO.	RANGE	MEAN
I	6	36- 64	50.3	4+1*	15- 51	36.8
II	23	72-168†	99.3	19	60-127	95.9
III‡	38	111-240	149.3	36	108-274	152.4
IV	2	262-288	275.0	1	------	254.0
Total	69	36-288	127.7	61	15-274	127.0

*In the case which is 15 months of age only $\frac{ba\ /a}{ba/\ a}$ are erupted.

†The case with age 168 months in stage II shows many teeth congenitally absent; excepting this the range is 72 to 132 months.

‡The average age for males with $\frac{7\text{-}1\ /1\text{-}7}{7\text{-}1/\ 1\text{-}7}$ erupted but no eruption of $\frac{8\ /8}{8/\ 8}$ is 138.0 months; for females 132.1; the numbers are 30 and 21, respectively. The remaining 8 males average 191.5 months and the 15 females 180.7 months.

The relation between occlusion and dentition stage (Table II) is not clear. There is the suggestion that malocclusion is less easily defined in stages I and early II than in the later divisions, and the implication is that some of the cases classified as normal in these periods would later be recorded as abnormal.

Table III presents the distribution of maxillary and mandibular arch forms according to dentition stage. The greater emphasis of ovoid and U-shaped arches in stages I and II clearly is evident. The tendency for the maxillary arch to emphasize tapering form and for the mandibular arch to show trapezoidal form is interesting. This is especially clear in females. Dentition stage IV is numerically too inadequate to be meaningful. Although the tendency

TABLE II

DENTITION STAGES AND OCCLUSION

DENTITION STAGE	MALE				FEMALE			
	NORMAL	I	II	III	NORMAL	I	II	III
I	4	1	1	0	1	3	1	0
II	3	6	10	4	2	8	8	1
III	3	21	13	1	3	19	11	3
IV	0	1	0	0	0	1	0	0
Totals	10	29	24	5	6	31	20	4

for ovoid and U-shaped arch forms to concentrate in stages I and II is clear, another fact is equally striking; children showing U-shaped and ovoid arch forms are found in 24 families; of these 24 families one, the other, or both parents present ovoid or U-shaped arches in 13 instances. Consequently the hereditary implications cannot be denied even at the early age levels.

TABLE III

DENTITION STAGES AND ARCH FORM

MALES						FEMALES					
A. MAXILLARY ARCH						A. MAXILLARY ARCH					
	TAPER	TRAPEZOID	OVOID	U-SHAPE	SQUARE		TAPER	TRAPEZOID	OVOID	U-SHAPE	SQUARE
I	1	0	4	1	0	I	0	1	2	1	0
II	9	3	4	6	0	II	5	5	4	2	0
III	11	10	4	5	1	III	17	11	4	1	0
IV	0	0	1	0	0	IV	0	0	0	1	0
Totals	21	13	13	12	1	Totals	22	17	10	5	0
B. MANDIBULAR ARCH						B. MANDIBULAR ARCH					
I	1	1	3	1	0	I	0	1	2	1	0
II	9	7	3	2	1	II	2	12	0	2	0
III	10	14	2	4	1	III	10	19	2	2	0
IV	0	0	1	0	0	IV	0	0	0	1	0
Totals	20	22	9	7	2	Totals	12	32	4	6	0

It is apparent that observations upon crowding do not attain a great deal of significance until the permanent dentition is moderately advanced in eruption (Table IV). The excess of mandibular over maxillary crowding is evident in both sexes.

TABLE IV

DENTITION STAGES AND CROWDING AND SPACING OF ANTERIORS

CROWDING AND SPACING DENTITION	MALES			FEMALES		
	SPACING	ABSENT	CROWDING	SPACING	ABSENT	CROWDING
A. MAXILLARY						
I	4	2	0	4	1	0
II	10	6	5	5	8	6
III	10	12	16	9	11	14
IV	1	1	0	1	0	0
Totals	25	21	21	19	20	20
B. MANDIBULAR						
I	4	2	0	3	2	0
II	1	10	10	2	10	7
III	3	13	22	4	12	19
IV	0	2	0	0	0	1
Totals	8	27	32	9	24	27

Our data fail to reveal that observations upon displacements (see Fig. 1) and asymmetries are much affected by age. Observations upon absolute size are based upon norms; hence while a great deal of development is shown in these factors, the concept of age is implicit in the size recorded. Age trends are but slightly reflected in our observations on obtusity or acuteness of mandibular angle and in depth of bite. These findings are rather contrary to expectation.

In summary then it is apparent that occlusion, arch form, and crowding and spacing of anteriors change considerably with age. The evidence for age effect upon other attributes is negative rather than conclusive in our present data.

GENETIC TRIANGLES

Before proceeding to the detailed analysis of the material it will be well to give a brief overview of the total data in order to provide a reference frame for particular features later on. The basis of this total analysis is the genetic triangle consisting of one mother and one father for each child. Thus statistically the number of parents will always be twice the number of children, or the number of female parents will equal the number of male parents and the number of male parents will equal the number of children. For purposes of this summary overview we have utilized observations upon displacements, inclination of maxillary and mandibular teeth, asymmetries, absolute size, angle between maxillary occlusal and Frankfort planes, obtusity or acuteness of mandibular angle, arch form, congenital absence of teeth, supernumerary teeth, malformed teeth, hypoplasia of enamel, crowding and spacing of teeth, depth of bite, crossbites, and classification of occlusion. It has seemed desirable to omit other observations listed in the recording blank (Figs. 1 and 2). The number of observations available in the genetic triangle analysis is recorded in Table V.

TABLE V

OBSERVATIONS IN GENETIC TRIANGLE ANALYSIS

NO. OF OBSERVATIONS	NO. OF FAMILIES	NO. OF CHILDREN	TOTAL NO. OBSERVATIONS
46	14	27	1242
36	28	45	1620
35	1	6	210
34	1	1	34
33	1	1	33
32	3	5	160
25	1	1	25
24	1	1	24
23	1	1	23
21	1	1	21
19	2	4	76
14	1	1	14
11	1	1	11
Totals	56	95	3493

The number of same observations made on all members of the family is shown in column 1; columns 2 and 3 record the number of families and the contained number of children, respectively; column 4 presents the total num-

ber of observations that may be arranged into genetic triangles. Thus we have 56 families with 95 children with the same observations available for all members of the family. This gives us a total of 3,493 observed features contained in 95 genetic triangles. We now wish (1) to contrast resemblance and not resemblance between parents, (2) to contrast resemblance and not resemblance between each child and each parent, (3) to contrast resemblance and not resemblance between each child and his two parents, when: (a) male parent and female parent are alike for the attribute, and (b) male parent and female parent are unlike for the attribute. Under the latter category some questions appear important:

1. Does the child resemble the male parent?
2. Does the child resemble the female parent?
3. Is the child intermediate between the two parents?
4. Does the child clearly show a feature that is vaguely presented in both parents? in the male parent? in the female parent?
5. Is the child unlike both parents?

In order to obtain a partial answer to these questions we legitimately may use the genetic triangle which statistically gives us 95 units with each one treated as a separate family even though the actuality is 95 children contained in 56 families. The first portion of the analysis is dependent upon two variables only; they can be the same or different. The degree of sameness or of differences does not concern us here. For example if we have recorded asymmetry of mandibular ramus height absent in mother, in father, and in child any relationship between the three members of the genetic triangle with reference to this feature must statistically be *same* or *resemblance* relationships. On the other hand if we have recorded the feature as absent in the father, questionably present on the right side in the mother, and questionably present on the left side in the child all relations in this genetic triangle must become *different* or *not resemblance* relationships. With only two choices available it is probable that random or chance factors will operate to produce 50 per cent of one variable and 50 per cent of the other;* consequently we become interested in percentage concentrations greater than 50 for interpretation. The results of this analysis are reproduced in Table VI. It is to be noted first that the amount of likeness between father and mother is 14.50 per cent in excess of the expected percentage. Many reasons, such as, general racial or national similarity, common circumstances in culture and environment, and so on, might be adduced to explain this excess. More important here is a purely negative answer. At least we cannot explain the excess in terms of genetics of the immediate family. The total amount of resemblance between father and child and between mother and child is somewhat higher than between father and mother but on the whole the difference (+3.08 per cent average) is not striking. There is little evidence to support the belief that children tend to resemble one parent more than they do the other; the percentage difference 0.93 is insignificant.

*Actually this is an oversimplification of the probabilities involved, but is deemed adequate for present purposes. For further discussion see Yule, G. Udney, *An Introduction to the Theory of Statistics*, Charles Griffin and Co., London, 1929, ed. 9, p. 33.

TABLE VI

RESEMBLANCE AND NONRESEMBLANCE

CATEGORY	%
1. Father like mother	64.50
2. Father unlike mother	35.50
3. Father like child	68.04
4. Father unlike child	31.96
5. Mother like child	67.11
6. Mother unlike child	32.89
7. Father and mother alike	
(a) child like parents	85.04
(b) child unlike parents	14.96
1. same feature opposite side	2.31
2. same feature emphasized	4.83
3. clearly different	7.82
8. Father and mother unlike	
(a) child like father	37.18
(b) child like mother	34.52
(c) child intermediate	16.94
(d) child different	11.36
1. clearly different	8.95
2. doubtfully different	2.41

The further analysis of resemblance between parents and child when mother and father are alike is shown in item 7 of the Table. The figures are striking; 85 per cent clearly are alike and only 8 per cent are clearly different. We do not interpret these figures to mean that 92 per cent of the traits we have examined in the dentofacial complex are to be ascribed to the hereditary mechanism, but we believe that they indicate genetic factors to be extensively operative.

The analysis of resemblance of child to parent or parents when father and mother are unlike is contained under item 8. Clear-cut differences not amenable to genetic interpretation obtain in 8.95 per cent of the features and, although the distribution of figures is somewhat different, the interpretation given for item 7 is generally substantiated. The percentage of intermediacy is unexpectedly low.

The presentation of the total data analyzed in terms of genetic triangles is intended as a brief overview. Rather extensive operation of hereditary factors in the dentofacial complex appears to be indicated. The further presentation is concerned with the analysis of specific features.

DETAILED ATTRIBUTES

All of the tables are arranged so that different parental combinations are shown in the vertical column at the left; the corresponding distribution of child traits is recorded in the respective horizontal lines. The number of families and the number of children involved for each parental combination are listed in the last two columns. The total distribution of child traits is shown in the last line.

ASYMMETRIES

The distribution of asymmetries is recorded in Table VII. All asymmetries are grouped into the categories present (p) and absent (a); specific types of asymmetry are not shown.

TABLE VII

ASYMMETRIES

PARENTS	CHILDREN PRESENT NO.	CHILDREN PRESENT %	CHILDREN ABSENT NO.	CHILDREN ABSENT %	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Ma Fa	2	14.3	12	86.7	9	14
Ma Fp	7	50.0	7	50.0	10	14
Mp Fa	8	28.6	20	71.4	15	28
Mp Fp	21	55.3	17	44.7	20	38
Ma Fu	7	33.3	14	66.7	12	21
Mp Fu	7	63.6	4	36.4	8	11
Total	52	41.3	74	58.7	74	126
Unobserved					4	4
Total					78	130

Observations were made on 74 families; these include 130 parents and 126 children. In 20 of these families, with 32 children, the father was unobserved (u).

One or more asymmetry is present in 41.3 per cent of children and 56.9 per cent of parents while 58.7 per cent of children and 43.1 per cent of parents show absence of facial asymmetry. In the parent groupings, however, 72 per cent show one, the other, or both parents with asymmetries and 28 per cent with an absence of asymmetry.

The detailed distribution reveals the number of asymmetries in children to rise markedly as parents show asymmetry. If we contrast the percentage of children showing asymmetry when both parents have an absence of asymmetry with those in which one, the other, or both parents present asymmetries, we find an average excess of 35.1 per cent attributable to the asymmetry of the parents. There is only one bracket in which a strong tendency for the operation of hereditary factors is contraindicated (mother present, father absent). Here we find 71.4 per cent of children showing asymmetries absent and 28.6 per cent showing asymmetries present. In all other instances the number of asymmetries is three to four times as great among children with asymmetrical parents as it is among children whose parents present an absence of asymmetry.

ABSOLUTE SIZE OF MAXILLA

Observations were made on 73 families and 124 children. The total distribution of absolute size in children, on the whole, is markedly symmetrical and highly concentrated in the category + (medium) size. There is a slightly higher percentage of deficient maxillas than of excess maxillas (9.7 per cent – 5.6 per cent). The difference is of doubtful statistical significance.

The part figures indicate a tendency of children to follow deficiency or excess in absolute size of maxilla if deficiency or excess is shown by parents. However, there are only two parental combinations in which the numbers are sufficiently large to warrant any conclusion: 1. Both parents + (medium); here there are 36 families with 64 children, and 82.8 per cent of the children are medium, 12.5 per cent are deficient (sm), and 4.7 per cent are excessive ++ in size. The tendency toward deficiency rather than excess is probably significant. 2. Mother medium, father unobserved; there are 17 families involving 26 children. The distribution is symmetrical and highly concentrated (92.4 per cent) in the medium category.

TABLE VIII

ABSOLUTE SIZE OF MAXILLA

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	0	0.0	1	100.0	0	0.0	1	1
M+ F+	8	12.5	53	82.8	3	4.7	36	64
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	2	25.0	6	75.0	0	0.0	4	8
M+ Fsm	0	0.0	3	100.0	0	0.0	2	3
M+ F++	0	0.0	7	87.5	1	12.5	5	8
M++ F+	0	0.0	5	83.3	1	16.7	4	6
Msm Fu	1	25.0	2	50.0	1	25.0	2	4
M+ Fu	1	3.8	24	92.4	1	3.8	17	26
M++ Fu	0	0.0	2	100.0	0	0.0	1	2
Total	12	9.7	105	84.7	7	5.6	73	124
Unobserved							5	6
Total							78	130

In two subgroups the mother presents a deficiency and the father is either medium or unobserved; the children present a 25.0 per cent deficiency. In 9 families involving 14 children, one parent presents an excess and the other is medium; 12 of the 14 children are medium; 2 present excesses, and none show deficiency.

The evidence for inheritance of deficiency or excess in the absolute size of the maxilla is not clear cut. There is, however, a tendency for children to follow the parental pattern. Since the majority of parents are medium (82.5 per cent), as regards absolute size of maxilla, it is to be presumed that most children would likewise be medium if hereditary factors were operative. The percentages are almost the same; 84.7 per cent of children are medium. Although there is question regarding inheritance of deficiency or excess, there seems little doubt that children will have medium size of maxilla if parents likewise have medium maxillary size.

ABSOLUTE SIZE OF MANDIBLE

Observations on the absolute size of the mandible were made on 73 families involving 124 children. The majority of children (75.0 per cent) and parents (74.6 per cent) are medium; deficiency is fairly common in children (23.4 per cent) and less frequent in parents (15.9 per cent), and excess is rare (1.6 per cent) in children and infrequent (9.5 per cent) in parents. The detailed distribution reveals a marked tendency for deficiency in mandibular size to occur in children when one or both parents present deficiency. In three of the four categories in which a parent presents deficiency, children likewise show deficiencies ranging between 40 per cent and 60 per cent. Equally noticeable, however, is the fact that deficiencies in children are not at all uncommon when both parents are medium or when one parent presents an excess and the other is medium. The percentage of excesses is too limited to warrant any conclusion. The negative evidence indicates that there is little tendency for mandibular excess to be transmitted.

Although there is a tendency of parental deficiencies to be transmitted to children, there is evidence that deficiency in the absolute size of the mandible

TABLE IX

ABSOLUTE SIZE OF MANDIBLE

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	3	60.0	2	40.0	0	0.0	3	5
M+ F+	10	23.8	32	76.2	0	0.0	31	42
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	4	40.0	6	60.0	0	0.0	4	10
M+ Fsm	3	18.8	13	81.2	0	0.0	5	16
M+ F++	2	22.2	7	78.8	0	0.0	6	9
M++ F+	0	0.0	8	100.0	0	0.0	3	8
Msm Fu	6	50.0	6	50.0	0	0.0	5	12
M+ Fu	1	5.3	17	89.4	1	5.3	14	19
M++ Fu	0	0.0	0	0.0	1	100.0	1	1
Total	29	23.4	93	75.0	2	1.6	73	124
Unobserved							5	6
Total							78	130

frequently obtains in children when contraindicated by parents. Aside from this piling up of deficiencies in children whose parents do not show deficiency, the evidence in general supports the conclusion that size factors are transmitted, at least in part, by the hereditary mechanism.

ABSOLUTE SIZE OF THE RAMUS

Observations were made upon 73 families involving 124 children. Ramus deficiencies are found in 50.8 per cent of cases and medium size of ramus obtains in 47.6 per cent. Excessive ramus size like excessive size of mandible is rare (1.6 per cent).

TABLE X

ABSOLUTE SIZE OF RAMUS

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	9	69.2	4	30.8	0	0.0	8	13
M+ F+	8	32.0	17	68.0	0	0.0	16	25
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	20	64.5	11	35.5	0	0.0	18	31
M+ Fsm	8	47.0	9	53.0	0	0.0	7	17
M+ F++	1	33.3	2	66.7	0	0.0	2	3
Msm F++	1	100.0	0	0.0	0	0.0	1	1
Msm Fu	11	78.6	3	21.4	0	0.0	8	14
M+ Fu	5	29.4	11	64.7	1	5.9	11	17
M++ Fu	0	0.0	0	0.0	1	100.0	1	1
Total	63	50.8	59	47.6	2	1.6	73	124
Unobserved							5	6
Total							78	130

The detailed figures show clearly a pronounced tendency for parents to transmit deficiency of ramus size to children. In the 5 parental combinations in which one, the other, or both parents have deficient rami, the children present deficiencies varying between 47.0 per cent and 100.0 per cent; in only one of the brackets does the percentage fall below 65.0. Of the remaining

parental combinations in which both parents are medium or one or both parents present an excess, the children show approximately 30.0 per cent deficiency and only 2 show excesses.

There is little doubt that hereditary factors are operative in the production of deficiency in the absolute size of the rami. There also is indicated a tendency toward deficiency beyond hereditary expectation.

ABSOLUTE SIZE OF BODY

Observations were made upon 73 families involving 124 children. The total distribution shows 76.6 per cent medium, 15.3 per cent deficient, and 8.1 per cent excess. The detailed distribution (Table XI) indicates clearly that absolute size of the body conforms to the parental pattern. The combination M+ F++ is an exception: 36.4 per cent of children show deficiency, 54.5 per cent are +, and 9.1 per cent present excess. There is a tendency for medium parents to produce deficiency more frequently than excess.

The size of mandibular body in children is dependent, to a considerable extent, on the size of the mandibular body in parents.

TABLE XI

ABSOLUTE SIZE OF BODY

PARENTS	CHILDREN SUBMEDIUM NO.	SUBMEDIUM %	MEDIUM NO.	MEDIUM %	LARGE NO.	LARGE %	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	2	100.0	0	0.0	0	0.0	1	2
M+ F+	6	9.7	55	88.7	1	1.6	35	62
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	0	0.0	1	100.0	0	0.0	1	1
M+ Fsm	0	0.0	4	100.0	0	0.0	3	4
M+ F++	4	36.4	6	54.5	1	9.1	8	11
M++ F+	0	0.0	6	60.0	4	40.0	4	10
Msm Fu	6	85.7	1	14.3	0	0.0	3	7
M+ Fu	1	4.2	20	83.3	3	12.5	16	24
M++ Fu	0	0.0	0	0.0	1	100.0	1	1
Total	19	15.3	95	76.6	10	8.1	73	124
Unobserved							5	6
Total							78	130

ABSOLUTE SIZE OF MALAR BONE

Observations were made on 73 families with 124 children. Here there are 87 per cent of children who are medium, 8 per cent who are deficient, and 5 per cent who have excess size of malar bones. The detailed distribution, with one exception (M+ F++), shows a pronounced tendency for children to follow the parental pattern.

ABSOLUTE SIZE OF PALATE HEIGHT

Observations were made on 69 families including 119 children. The majority of parents (78.2 per cent) and children (80.7 per cent) present medium height of palate, low palates are found in 0.8 per cent of parents and 4.2 per cent of children, and high palates appear in 21.0 per cent of parents and 15.1

TABLE XII

ABSOLUTE SIZE OF MALAR BONE

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Msm Fsm	0	0.0	1	100.0	0	0.0	1	1
M+ F+	2	3.7	51	94.4	1	1.9	31	54
M++ F++	0	0.0	2	100.0	0	0.0	1	2
Msm F+	3	75.0	1	25.0	0	0.0	2	4
M+ Fsm	1	10.0	9	90.0	0	0.0	5	10
M+ F++	2	15.4	9	69.2	2	15.4	8	13
M++ F+	0	0.0	5	83.3	1	16.7	3	6
M++ Fsm	0	0.0	2	100.0	0	0.0	2	2
Msm Fu	1	25.0	3	75.0	0	0.0	2	4
M+ Fu	1	3.8	24	92.4	1	3.8	17	26
M++ Fu	0	0.0	1	50.0	1	50.0	1	2
Total	10	8.1	108	87.1	6	4.8	73	124
Unobserved							5	6
Total							78	130

per cent of children. The detailed distribution clearly shows children with high palates to have one, the other, or both parents with excessive height of palate (Table XIII). The number of parents and children with low palates is too small to warrant conclusion.

TABLE XIII

ABSOLUTE SIZE OF PALATE HEIGHT

PARENTS	CHILDREN SUBMEDIUM NO.	%	MEDIUM NO.	%	LARGE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
M+ F+	1	2.0	40	93.0	2	4.7	26	43
M++ F++	0	0.0	6	60.0	4	40.0	5	10
M+ Fsm	0	0.0	2	100.0	0	0.0	1	2
M+ F++	1	4.5	17	77.3	4	18.2	12	22
M++ F+	1	7.7	8	61.5	4	30.8	7	13
M+ Fu	2	8.7	18	78.3	3	13.0	14	23
M++ Fu	0	0.0	5	83.3	1	16.7	4	6
Total	5	4.2	96	80.7	18	15.1	69	119
Unobserved							9	11
Total							78	130

Hereditary factors are operative in the production of normal and excessive height of palate.

ABSOLUTE SIZE OF PALATE WIDTH

Observations were made upon 67 families involving 117 children. The majority of children (65.8 per cent) are medium in width, approximately one-fourth (23.9 per cent) are deficient, and the remainder (10.3 per cent) show excessive width. The parents present more medium (76.7 per cent) and fewer deficient (16.4 per cent) and excess (6.9 per cent) palate widths than children. The subgroup figures indicate, on the whole, deficiency and excess to be found when indicated by parent combination; both parents medium (M+ F+) is a notable exception with 29.8 per cent of children presenting deficiency.

Although the evidence is not clear cut, there is indication, on the whole, that hereditary factors play an important role in the width of palate.

TABLE XIV

ABSOLUTE SIZE OF PALATE WIDTH

PARENTS	CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
	SUBMEDIUM		MEDIUM		LARGE			
	NO.	%	NO.	%	NO.	%		
Msm Fsm	0	0.0	3	75.0	1	25.0	2	4
M+ F+	14	29.8	30	63.8	3	6.4	28	47
Msm F+	6	35.3	9	52.9	2	11.8	9	17
M+ Fsm	5	71.4	2	28.6	0	0.0	3	7
M+ F++	0	0.0	5	71.4	2	28.6	4	7
M++ F+	0	0.0	5	83.3	1	16.7	3	6
Msm Fu	3	42.9	3	42.9	1	14.2	3	7
M+ Fu	0	0.0	19	90.5	2	9.5	14	21
M++ Fu	0	0.0	1	100.0	0	0.0	1	1
Total	28	23.9	77	65.8	12	10.3	67	117
Unobserved							11	13
Total							78	130

OBTUSITY OF ANGLE

Observations on the obtusity and acuity of the mandibular angle were made on 70 families involving 120 children. The total figures show that a normal degree of obtusity (obtusity absent, obtusity?) is found in 56 per cent of cases and that somewhat marked angular obtusity (+, ++) is found in 43 per cent of the cases. Acuity of the mandibular angle is rare in children, being found in only one of the cases (0.8 per cent).

The detailed distribution reveals clearly that angular obtusity, on the whole, is found in children when one, the other, or both parents present this feature, and that absence of obtusity in children follows the parental pattern (Table XV).

The shape of the mandibular angle is dependent primarily upon hereditary factors.

MAXILLARY ARCH FORM

Observations were made upon 54 families involving 99 children. The total distribution reveals that tapering and trapezoidal arch forms are predominant in children (62.5 per cent), and that these forms are also most common in parents—42 of the 54 families present one, the other, or both parents with a tapering or trapezoidal maxillary arch. Ovoid arches are found in 21.2 per cent of children, U-shaped arches are found in 15.1 per cent, and square arches are rare. The percentage of ovoid arch forms is rather low considering the fact that several children present complete or almost complete deciduous dentitions.

The detailed distribution reveals a strong hereditary tendency in the inheritance of arch form. In addition to the tendency for children to follow the general parental pattern of arch form, there is a rather marked tendency for the maxillary arch to show constriction (tapering and trapezoidal forms) with a greater frequency than would be expected from the various parental combinations.

The shape of the maxillary arch is strongly conditioned by hereditary factors and constriction appears more frequently than expected.

TABLE XV

OBTUSITY OF ANGLE

PARENTS		CHILDREN SHOWING ABSENCE		OBTUSITY ?		OBTUSITY +		OBTUSITY ++		ACUITY ?		NUMBER OF FAMILIES	NUMBER OF CHILDREN
		NO.	%	NO.	%	NO.	%	NO.	%	NO.	%		
Ma	Fa	2	66.7	1	33.3	0	0.0	0	0.0	0	0.0	3	3
M ?	Fa	2	20.0	8	80.0	0	0.0	0	0.0	0	0.0	5	10
Ma	F ?	3	50.0	3	50.0	0	0.0	0	0.0	0	0.0	3	6
M +	F +	0	0.0	1	8.3	10	83.3	1	8.3	0	0.0	8	12
M +	Fa	4	40.0	3	30.0	3	30.0	0	0.0	0	0.0	6	10
Ma	F +	0	0.0	0	0.0	0	0.0	1	100.0	0	0.0	1	1
M ++	F ++	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
M ++	Fa	3	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	3
M +	F ?	1	50.0	0	0.0	1	50.0	0	0.0	0	0.0	1	2
M ?	F +	3	25.0	1	8.3	8	66.7	0	0.0	0	0.0	7	12
M ++	F ?	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	1	1
M ?	F ++	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
M ++	F +	1	33.3	0	0.0	2	66.7	0	0.0	0	0.0	1	3
Ma	Fu	6	42.9	4	28.6	3	21.4	1	7.1	0	0.0	8	14
M ?	Fu	0	0.0	2	50.0	2	50.0	0	0.0	0	0.0	4	4
M +	Fu	1	7.1	4	28.6	9	64.3	0	0.0	0	0.0	8	14
Mac +	Fac +	0	0.0	1	50.0	1	50.0	0	0.0	0	0.0	1	2
Mac ++	Fac ++	1	50.0	0	0.0	0	0.0	0	0.0	1	50.0	1	2
Mob +	Fac +	2	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	2
Mac ?	Fob ?	1	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	1
M ?	F ?	0	0.0	7	43.7	9	56.3	0	0.0	0	0.0	7	16
Total		30	25.0	37	30.8	49	40.8	3	2.5	1	0.8	70	120
Unobserved												8	10
Total												78	130

TABLE XVI

MAXILLARY ARCH FORM

PARENTS		CHILDREN SHOWING TAPERING		TRAPEZOIDAL		U-SHAPED		SQUARE		OVOID		NUMBER OF FAMILIES	NUMBER OF CHILDREN
		NO.	%	NO.	%	NO.	%	NO.	%	NO.	%		
Mta	Fta	3	42.8	3	42.8	0	0.0	0	0.0	1	14.2	5	7
Mtr	Ftr	5	35.7	4	28.6	2	14.3	0	0.0	3	21.4	9	14
Mu	Fu	1	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	1
Mov	Fov	1	14.3	1	14.3	1	14.3	0	0.0	4	57.1	3	7
Mtr	Fta	2	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	2
Mov	Fta	0	0.0	0	0.0	0	0.0	0	0.0	1	100.0	1	1
Mta	Ftr	8	47.1	4	23.5	2	11.8	0	0.0	3	17.6	8	17
Mu	Ftr	2	66.7	1	33.3	0	0.0	0	0.0	0	0.0	2	3
Mov	Ftr	4	66.7	1	16.7	1	16.7	0	0.0	0	0.0	2	6
Mta	Fu	0	0.0	3	60.0	2	40.0	0	0.0	0	0.0	2	5
Mtr	Fu	0	0.0	0	0.0	0	0.0	1	100.0	0	0.0	1	1
Mov	Fu	1	33.3	0	0.0	2	66.7	0	0.0	0	0.0	1	3
Mu	Fov	1	50.0	0	0.0	0	0.0	0	0.0	1	50.0	2	2
Mov	Fsq	0	0.0	1	50.0	0	0.0	0	0.0	1	50.0	1	2
Mta	Fov	2	100.0	0	0.0	0	0.0	0	0.0	0	0.0	1	2
Mtr	Fov	0	0.0	0	0.0	1	50.0	0	0.0	1	50.0	1	2
Mta	Fun	4	44.4	0	0.0	1	11.1	0	0.0	4	44.4	3	9
Mtr	Fun	2	22.2	6	66.7	0	0.0	0	0.0	1	11.1	7	9
Mu	Fun	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	1	1
Mov	Fun	2	40.0	0	0.0	2	40.0	0	0.0	1	20.0	2	5
Total		38	38.3	24	24.2	15	15.1	1	1.0	21	21.2	54	99
Unobserved												24	31
Total												78	130

TABLE XVII

MANDIBULAR ARCH FORM

PARENTS		CHILDREN SHOWING TAPERING		TRAPEZOIDAL		U-SHAPED		SQUARE		OVOID		NUMBER OF FAMILIES	NUMBER OF CHILDREN
		NO.	%	NO.	%	NO.	%	NO.	%	NO.	%		
Mta	Fta	4	44.4	4	44.4	1	11.1	0	0.0	0	0.0	4	9
Mtr	Ftr	7	28.0	12	48.0	2	8.0	1	4.0	3	12.0	13	25
Mu	Fu	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
Mov	Fov	0	0.0	0	0.0	2	50.0	0	0.0	2	50.0	2	4
Mta	Ftr	2	20.0	8	80.0	0	0.0	0	0.0	0	0.0	6	10
Mu	Ftr	2	66.7	1	33.3	0	0.0	0	0.0	0	0.0	2	3
Mov	Ftr	0	0.0	3	100.0	0	0.0	0	0.0	0	0.0	2	3
Mov	Fta	1	33.3	1	33.3	0	0.0	0	0.0	1	33.3	1	3
Mtr	Fta	2	40.0	2	40.0	0	0.0	0	0.0	1	20.0	4	5
Mu	Fov	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	1	1
Mov	Fsq	0	0.0	2	100.0	0	0.0	0	0.0	0	0.0	1	2
Mtr	Fu	0	0.0	3	75.0	1	25.0	0	0.0	0	0.0	2	4
Mov	Fu	1	33.3	0	0.0	2	66.7	0	0.0	0	0.0	1	3
Mtr	Fov	0	0.0	2	100.0	0	0.0	0	0.0	0	0.0	1	2
Mta	Fun	2	28.6	1	14.3	1	14.3	0	0.0	3	42.8	2	7
Mtr	Fun	3	23.1	8	61.5	0	0.0	0	0.0	2	15.4	9	13
Mu	Fun	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
Mov	Fun	0	0.0	1	33.3	1	33.3	0	0.0	1	33.3	1	3
Total		24	24.2	50	50.5	11	11.1	1	1.0	13	13.1	54	99
Unobserved												24	31
Total												78	130

MANDIBULAR ARCH FORM

Observations were made upon 54 families with 99 children. Tapering and trapezoidal arches are predominant (74.7 per cent), square arches are rare (1.0 per cent), hyperbolic arches are unrepresented, U-shaped arches (11.1 per cent) and ovoid arch forms (13.1 per cent) are moderately frequent (Table XVII).

As in maxillary arch form, there is a strong tendency for the mandibular arch form to follow an hereditary pattern; the dominance of constricted arch forms (tapering and trapezoidal) is indicated.

Arch form is largely hereditary and dominance of constricted over non-constricted types is indicated.

CONGENITAL ABSENCE OF TEETH

Observations were made upon 67 families involving 113 children. The majority of parents and children observed do not have congenitally absent teeth. A sizable percentage (15.0 per cent), however, do present congenital absence of teeth.

The detailed distribution table according to parental pattern clearly shows the hereditary nature of congenital absence of teeth.

TABLE XVIII

CONGENITAL ABSENCE OF TEETH

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	4	5.6	67	94.4	42	71
Mp	Fp	7	63.6	4	36.4	6	11
Ma	Fp	2	66.7	1	33.3	2	3
Ma	Fu	1	4.5	21	95.5	13	22
Mp	Fu	3	50.0	3	50.0	4	6
Total		17	15.0	96	85.0	67	113
Unobserved						11	17
Total						78	130

SUPERNUMERARY TEETH

Observations were made upon 66 families including 111 children. The total distribution shows supernumerary teeth largely are absent in the parents and children of this series. Although the evidence is too limited to permit conclusion, the operation of hereditary factors is suggested.

TABLE XIX

SUPERNUMERARY TEETH

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	1	1.3	77	98.7	47	78
Ma	Fp	1	20.0	4	80.0	2	5
Ma	Fu	1	3.6	27	96.4	17	28
Total		3	2.7	108	97.3	66	111
Unobserved						12	19
Total						78	130

MALFORMED TEETH

Observations were made upon 67 families with 113 children. Malformed teeth are uncommon both in the parents and in the children constituting this series. What evidence there is does not reveal the operation of hereditary factors.

TABLE XX

MALFORMED TEETH

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	5	6.2	76	93.8	48	81
Ma	Fp	0	0.0	2	100.0	1	2
Mp	Fp	0	0.0	2	100.0	1	2
Ma	Fu	1	3.6	27	96.4	17	28
Total		6	5.3	107	94.7	67	113
Unobserved						11	17
Total						78	130

HYPOPLASIA OF ENAMEL

Observations were made upon 67 families and 113 children. Hypoplasia of enamel is uncommon both in parents and in children. There is no evidence, although the data are very limited, pointing toward the inheritance of enamel hypoplasia.

TABLE XXI

HYPOPLASIA OF ENAMEL

PARENTS		CHILDREN				NUMBER OF FAMILIES	NUMBER OF CHILDREN
		PRESENCE		ABSENCE			
		NO.	%	NO.	%		
Ma	Fa	5	5.9	80	94.1	50	85
Ma	Fu	0	0.0	27	100.0	16	27
Ma	Fu	0	0.0	1	100.0	1	1
Total		5	4.4	108	95.6	67	113
Unobserved						11	17
Total						78	130

CROWDING AND SPACING OF MAXILLARY ANTERIORS

Observations were made upon 64 families with 109 children. Spacing, crowding, and absence of either spacing or crowding are equally distributed in the total group of children. Absence of crowding or spacing is found in 61.8 per cent of the parents, crowding in 26.4 per cent, and spacing in 11.8 per cent. The detailed distribution indicates that the children, on the whole, follow the parental pattern (Table XXII). Crowding or spacing of maxillary anteriors seems to be dependent upon a number of factors, as both crowding and spacing appear in moderately large percentages (25.0 per cent, 33.3 per cent) when neither parent shows crowding or spacing. Likewise, the distribution of crowding and spacing in children when one, the other, or both parents show crowding or spacing or some combination of these two, indicates multiple factors to be involved.

TABLE XXII

CROWDING AND SPACING OF MAXILLARY ANTERIORS

PARENTS		CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
		SPACING		ABSENCE		CROWDING			
		NO.	%	NO.	%	NO.	%		
Ma	Fa	8	33.3	10	41.7	6	25.0	17	24
Mp	Fp	0	0.0	3	50.0	3	50.0	4	6
Ma	Fp	3	15.0	6	30.0	11	55.0	10	20
Mp	Fa	1	12.5	2	25.0	5	62.5	5	8
Msp	Fa	8	57.1	5	35.7	1	7.1	5	14
Ma	Fsp	3	50.0	2	33.3	1	16.7	4	6
Msp	Fsp	0	0.0	0	0.0	1	100.0	1	1
Ma	Fu	10	52.6	5	26.3	4	21.1	10	19
Mcr	Fu	2	22.2	3	33.3	4	44.4	6	9
Msp	Fu	1	50.0	0	0.0	1	50.0	2	2
Total		36	33.0	36	33.0	37	33.9	64	109
Unobserved								14	21
Total								78	130

Hereditary factors (probably multiple) are operative in producing crowding or spacing of maxillary anterior teeth. The factor of age, as previously shown, is also involved.

CROWDING AND SPACING OF MANDIBULAR ANTERIORS

Observations were made upon 67 families involving 114 children. Crowding of mandibular anteriors among children occurs more frequently (47.4 per cent), than spacing (10.5 per cent), or absence of crowding or spacing (42.1 per cent). Parents present a slightly higher percentage of crowding (49.1 per cent), a lower incidence of spacing (2.5 per cent), and a greater amount of absence of crowding or spacing (48.3 per cent) than children. The differences are of doubtful significance. Children follow the parental pattern to a considerable extent. Crowding of mandibular anteriors is somewhat more frequent than would be expected from the parental combination. Possibly partial dominance of factors controlling crowding is involved. Multiple factors may be operative.

TABLE XXIII

CROWDING AND SPACING OF MANDIBULAR ANTERIORS

PARENTS		CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
		SPACING		ABSENCE		CROWDING			
		NO.	%	NO.	%	NO.	%		
Ma	Fa	3	15.8	11	57.9	5	26.3	14	19
Mp	Fp	3	8.6	12	34.3	20	57.1	15	35
Ma	Fp	0	0.0	6	37.5	10	62.5	10	16
Mp	Fa	1	9.1	4	36.4	6	54.5	8	11
Msp	Fa	0	0.0	1	100.0	0	0.0	1	1
Msp	Fcr	0	0.0	0	0.0	2	100.0	1	2
Ma	Fu	4	22.2	10	55.6	4	22.2	10	18
Mcr	Fu	0	0.0	4	40.0	6	60.0	7	10
Msp	Fu	1	50.0	0	0.0	1	50.0	1	2
Total		12	10.5	48	42.1	54	47.4	67	114
Unobserved								11	16
Total								78	130

The hereditary mechanism operates, at least in part, to produce crowding or spacing of mandibular anterior teeth. Partial dominance or multiple factors may be involved.

CROWDING AND SPACING OF MAXILLARY BUCCAL TEETH (TABLE XXIV)

Observations were made upon 61 families involving 105 children. Crowding of the maxillary buccal teeth is relatively rare in children and in parents. The total distribution for parents and children is symmetrical and highly concentrated in the absent category. There is little indication that heredity operates in crowding and spacing of maxillary buccal teeth, except for negative evidence in the parental combinations mother absent, father absent and mother absent, and father unobserved.

The numbers in the other categories are too limited to warrant any conclusion, but the trend is away from the operation of hereditary factors.

TABLE XXIV

CROWDING AND SPACING OF MAXILLARY BUCCAL TEETH

PARENTS		CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
		SPACING		ABSENCE		CROWDING			
		NO.	%	NO.	%	NO.	%		
Ma	Fa	2	3.1	62	96.8	0	0.0	36	64
Mp	Fp	0	0.0	2	100.0	0	0.0	1	2
Ma	Fp	1	25.0	3	75.0	0	0.0	3	4
Mp	Fa	0	0.0	4	80.0	1	20.0	3	5
Ma	Fu	0	0.0	26	96.3	1	3.7	16	27
Mcr	Fu	0	0.0	3	100.0	0	0.0	2	3
Total		3	2.8	100	95.2	2	2.0	61	105
Unobserved								17	25
Total								78	130

CROWDING AND SPACING OF MANDIBULAR BUCCAL TEETH (TABLE XXV)

Observations were made upon 63 families involving 107 children. Crowding or spacing of mandibular buccal teeth are relatively rare, both in children (12.9 per cent) and in parents (4.6 per cent). The detailed figures do not reveal the operation of hereditary factors as regards crowding or spacing of mandibular buccal teeth.

TABLE XXV

CROWDING AND SPACING OF MANDIBULAR BUCCAL TEETH

PARENTS		CHILDREN						NUMBER OF FAMILIES	NUMBER OF CHILDREN
		SPACING		ABSENCE		CROWDING			
		NO.	%	NO.	%	NO.	%		
Ma	Fa	3	4.2	64	88.9	5	6.9	41	72
Ma	Fp	0	0.0	2	100.0	0	0.0	2	2
Mp	Fa	0	0.0	3	75.0	1	25.0	2	4
Ma	Fun	2	7.1	23	82.1	3	10.7	17	28
Mcr	Fun	0	0.0	2	100.0	0	0.0	1	2
Total		5	4.6	94	87.0	9	8.3	63	108
Unobserved								15	22
Total								78	130

ROTATED TEETH

Observations were made upon 65 families with 111 children. Markedly rotated teeth are found in approximately half both of parents (50.9 per cent)

and of children (47.4 per cent). The detailed distribution clearly demonstrates the operation of hereditary factors, apparently single genes segregating normally.

TABLE XXVI

ROTATED TEETH

PARENTS		CHILDREN PRESENCE NO.	%	ABSENCE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Ma	Fa	4	21.1	15	78.9	14	19
Mp	Fp	25	80.6	6	19.4	15	31
Ma	Fp	8	44.4	10	55.6	11	18
Mp	Fa	8	50.0	8	50.0	9	16
Ma	Fu	1	7.1	13	92.9	8	14
Mp	Fu	7	53.8	6	46.2	8	13
Total		53	47.7	58	52.3	65	111
Unobserved						13	19
Total						78	130

DEPTH OF BITE

Observations were made upon 62 families including 105 children. Pronounced to extreme (++, +++) depth of bite is found in 62.8 per cent of children and in 46.7 per cent of parents. Extreme depth of bite is more frequent in children (15.2 per cent) than in parents (11.2 per cent). Absent and medium depth of bite occur in 30.4 per cent of children and in 52.3 per cent of parents. Open-bite is rare in parents (0.9 per cent) and infrequent in children (6.7 per cent). The detailed distribution (Table XXVII) fails to show the operation of hereditary factors in the production of open-bite; evidence, however, is very limited but does show clearly that deep bites are considerably dependent upon the presence of deep bites in one, the other, or both parents. In general, it seems that children show a somewhat more pronounced depth of bite than would be expected from the parental combination.

ANTERIORS IN CROSSBITE

Observations were made upon 65 families involving 109 children. Anterior teeth in crossbite are found in 14.7 per cent of children and in 9.8 per cent of parents. The operation of hereditary factors in the production of anteriors in crossbite, on the whole, is contraindicated by the detailed distribution.

OTHER CROSSBITES

Observations were made upon 65 families involving 108 children. The total distribution shows 13 per cent of the children present other crossbites, and 87 per cent show other crossbites absent. The detailed distribution fails to show the operation of hereditary factors, with reference to buccal teeth in crossbite (Table XXIX).

OCCLUSION

Observations were made upon 66 families involving 110 children. The total distribution, as would be expected from the type of sample, shows the majority of children to have some type of malocclusion. Of the children, 49.1

TABLE XXVII

DEPTH OF BITE

PARENTS		CHILDREN SHOWING ABSENCE		MEDIUM		PRONOUNCED		EXTREME		OPEN +		OPEN ++		NO. OF FAMILIES	NO. OF CHILDREN
		NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%		
Ma	Fa	0	0.0	0	0.0	1	50.0	0	0.0	1	50.0	0	0.0	2	2
M +	F +	0	0.0	1	9.1	7	63.6	2	18.2	1	9.1	0	0.0	5	11
M ++	F ++	4	21.0	4	21.0	9	47.4	1	5.3	1	5.3	0	0.0	10	19
M +++	F +++	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
Ma	F +	1	33.3	0	0.0	2	66.7	0	0.0	0	0.0	0	0.0	2	3
M +	Fa	1	25.0	2	50.0	1	25.0	0	0.0	0	0.0	0	0.0	4	4
M ++	F +	0	0.0	1	12.5	4	50.0	3	37.5	0	0.0	0	0.0	6	8
M +	F ++	0	0.0	1	16.7	2	33.3	2	33.3	0	0.0	1	16.7	4	6
Ma	F ++	4	57.1	1	14.3	0	0.0	2	28.6	0	0.0	0	0.0	4	7
Ma	F +++	0	0.0	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	1	1
M +	F +++	0	0.0	0	0.0	1	50.0	1	50.0	0	0.0	0	0.0	1	2
M +++	F +	0	0.0	0	0.0	1	100.0	0	0.0	0	0.0	0	0.0	1	1
M +++	Fa	0	0.0	3	50.0	2	33.3	1	16.7	0	0.0	0	0.0	1	6
M ++	F +++	1	20.0	0	0.0	2	40.0	2	40.0	0	0.0	0	0.0	3	5
Ma	Fun	2	66.7	0	0.0	0	0.0	0	0.0	1	33.3	0	0.0	2	3
M +	Fun	1	7.7	3	23.1	8	61.5	0	0.0	1	7.7	0	0.0	10	13
M ++	Fun	0	0.0	1	16.6	5	83.3	0	0.0	0	0.0	0	0.0	3	6
M +++	Fun	0	0.0	0	0.0	4	80.0	1	20.0	0	0.0	0	0.0	1	5
Mop +	Fun	0	0.0	1	50.0	0	0.0	0	0.0	1	50.0	0	0.0	1	2
Total		14	13.3	18	17.1	50	47.6	16	15.2	6	5.7	1	1.0	62	105
Unobserved														16	25
Total														78	130

TABLE XXVIII

ANTERIORS IN CROSSBITE

PARENTS		CHILDREN PRESENCE NO.	%	ABSENCE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Ma	Fa	10	15.2	56	84.8	38	66
Ma	Fp	1	14.3	6	85.7	5	7
Mp	Fa	2	33.3	4	66.7	3	6
Mp	Fp	0	0.0	1	100.0	1	1
Ma	Fu	3	11.1	24	88.9	17	27
Mp	Fu	0	0.0	2	100.0	1	2
Total		16	14.7	93	85.3	65	109
Unobserved						13	21
Total						78	130

TABLE XXIX

OTHER CROSSBITES

PARENTS		CHILDREN PRESENCE NO.	%	ABSENCE NO.	%	NUMBER OF FAMILIES	NUMBER OF CHILDREN
Ma	Fa	9	15.5	49	84.5	33	58
Ma	Fp	1	12.5	7	87.5	6	8
Mp	Fa	1	9.1	10	90.9	6	11
Mp	Fp	0	0.0	2	100.0	1	2
Ma	Fu	3	12.0	22	88.0	17	25
Mp	Fu	0	0.0	4	100.0	2	4
Total		14	13.0	94	87.0	65	108
Unobserved						13	22
Total						78	130

per cent show Class I, 32.7 per cent show Class II, 6.4 per cent show Class III, and 11.8 per cent of the children are normal or borderline normal. The majority of parents (64.8 per cent) are Class I. Here the malocclusion is associated with mutilation in many instances. Class II malocclusions rank second among parents (21.3 per cent), normal occlusion obtains in 9.8 per cent and Class III is found in 4.1 per cent of parents.

The detailed distribution indicates clearly that hereditary factors are involved in the production of malocclusion, although the patterning according to occlusion type is not clear. It is to be noted that the majority of those children showing normal occlusions in this series, present at least one parent with a normal occlusion and with the other parent tending to show a Class I malocclusion.

Children in this series present a high incidence of malocclusion. This is to be expected since the basis of selection of the sample is the presence of a malocclusion in at least one child in each family represented. There is evidently an operation of hereditary factors in the production of malocclusion but the patterning is not clear. It is more probable that occlusion is dependent upon many traits, each of which is dependent to a greater or lesser degree upon hereditary factors as indicated in the previous sections of the discussion.

DIAGNOSIS AND TREATMENT

Two points have been brought out in the analysis which warrant further consideration from the standpoint of the bearing they have on diagnosis and

TABLE XXX

OCCLUSION

PARENTS		CHILDREN SHOWING NORMALCY		CHILDREN SHOWING CLASS I		CHILDREN SHOWING CLASS II		CHILDREN SHOWING CLASS III		NUMBER OF FAMILIES	NUMBER OF CHILDREN
		NO.	%	NO.	%	NO.	%	NO.	%		
Mn	Fn	0	0.0	0	0.0	0	0.0	1	100.0	1	1
Mn	FI	1	11.1	5	55.6	3	33.3	0	0.0	6	9
MI	Fn	1	50.0	1	50.0	0	0.0	0	0.0	1	2
MI	FI	2	5.6	24	66.7	9	25.0	1	2.8	22	36
MI	FII	2	20.0	4	40.0	4	40.0	0	0.0	5	10
MII	Fn	1	25.0	0	0.0	3	75.0	0	0.0	3	4
MII	FI	0	0.0	4	57.1	3	42.9	0	0.0	2	7
MII	FII	0	0.0	4	80.0	1	20.0	0	0.0	4	5
MI	FIII	0	0.0	0	0.0	0	0.0	1	100.0	1	1
MII	FIII	0	0.0	0	0.0	1	100.0	0	0.0	1	1
MIII	FI	2	66.7	0	0.0	0	0.0	1	33.3	1	3
MIII	FIII	0	0.0	0	0.0	1	100.0	0	0.0	1	1
Mn	Fun	0	0.0	0	0.0	0	0.0	1	100.0	1	1
MI	Fun	1	6.3	9	56.3	5	31.3	1	6.3	11	16
MII	Fun	3	23.1	3	23.1	6	46.2	1	7.7	6	13
Total		13	11.8	54	49.1	36	32.7	7	6.4	66	110
Unobserved										12	20
Total										78	130

treatment. The first of these is the extensive operation of heredity in the production of features in the dentofacial complex. If we assume—and we seem obliged to do so—that an individual is developing along the lines of his biologic expectation when he gives full expression to the hereditary circumstances that are initially responsible for him, we must recognize that many undesirable traits biologically are fully normal. We can no longer retain the point of view, "Nature never makes a mistake," with the subsequent assignment of all irregularities and anomalies to nurtural, developmental, or mechanical interferences with nature. And we must recognize that orthodontic procedure will be outlined on a policy of judicious interference with nature as frequently as it will be formulated on a basis of cooperation with nature. Probably the most important contribution hereditary knowledge makes to diagnosis is in the outlining of expectation for the growing child, especially in the bracket of attributes which are markedly influenced by age. It is pertinent to cite some examples. The arch form of the young child with a deciduous dentition characteristically is ovoid both above and below. There is some variability in the deciduous arch form and we find a few cases of markedly tapering arches as well as a few examples of hyperbolic arches. The majority, however, are more or less ovoid. While we have reasonable evidence that the tapering deciduous arch predicts a probably more extremely tapered adult arch, we can know little what the deciduous ovoid arch predicts without recourse to other evidence. We may do two things. The first is accurate: wait until the adult arch form has been observed before describing it. This ordinarily means that we must wait until the child is from 10 to 13 years old before we can make accurate appraisal. The second method is to estimate what the arch form will be through use of associated facts which permit useful generalizations. In arch form heredity provides this body of facts. We note that arch form is strongly dependent upon hereditary circumstances with a general dominance of narrowed or constricted arches

over broad ones. Thus if we know whether one, the other, or both parents present constricted arches, we are provided with a considerable body of evidence about the probable course of development in the child. In a like manner the other hereditary evidence in the dentofacial complex, as size and placement of the various parts of the mandible and maxilla, depth of bite, anterior crowding of teeth, etc., serves further to define the probable course of development of the child. We believe precision in estimate of growth sequences and adult configurations in the dentofacial complex to be fundamental in etiology and diagnosis of malocclusion. Further it appears axiomatic to us that a treatment procedure should be based upon etiologic and diagnostic evidence of the individual case.

The second point to be considered is the nature of a malocclusion or, better, of occlusion. Occlusion cannot be classified as an attribute; it is a configuration of many attributes. At the moment, we are not in a position to give an estimate of the number of attributes in the dentofacial complex. We know there are many rather than few and that the number of combinatorial groupings approaches astronomical figures. We estimate that half, or more, of the twenty-four pairs of human chromosomes are represented in the area with the resultant probability of a high degree of attribute independence. This occlusion, by definition a relationship phenomenon, is established largely by the probability circumstances which juxtapose the attributes of the dentofacial complex with one another. The size of the teeth may or may not conform to the amount of containing alveolus. The alveoli present but low order covariation with their supporting maxillary and mandibular bases. The mandible is far from an integrated whole; its several parts—condylar and coronoid processes, ascending rami, angles, body, alveolar process, and individual teeth —show a fairly high degree of independent variation. This same type of part independence is evident in the maxillary complex, and certainly, the maxillary and mandibular complexes are independent of one another.

The variations within and between the several parts of the dentofacial complex have a strong foundation in heredity; consequently an understanding of the operation of these phenomena is important in outlining treatment procedures. Somewhat as an aside may be added that nongenetic variability frequently is present. There is no doubt that the continued practice of oral habits, as thumb-sucking or cheek-biting, or mutilation of the dentition through extensive operation of caries or premature loss of teeth is orthodontically undesirable. It would be folly of the highest order to omit a consideration of environmental or nurtural circumstances in seeking facts about occlusion. Nor can one view without alarm the widespread laissez-faire attitude that the dentition and supporting structures are self-readjusting to all kinds of variations which are imposed by nonhereditary forces. Our sample fails to provide much evidence for the incidence of environmental variations of the above kinds for the total population. In our group hereditary variations are far more frequent than nonhereditary ones and the bulk of treatment cases have their primary etiology in attributes of the former type. These are particularly striking in Class II and Class III cases of all kinds and in Class I cases involving constriction in the anterior part of the arch and crowding of maxillary and mandibular anterior teeth.

The literature has made frequent reference to the proposition that a malocclusion which had a genetic foundation was not amenable to treatment or, if it were, the treatment would be more difficult and the frequency of relapse higher than if nongenetic factors only were responsible for the malocclusion. The proposition has some truth in it but on the whole, it is not accurate enough to warrant an unqualified recommendation to the orthodontic profession. Any structure may be modified within greater or lesser limits and difficulty usually is encountered when there is an attempt to exceed these limits. In the dentofacial complex there is considerable difference in the modification limits of the various parts. The individual teeth present probably the narrowest limits; their size, shape, number, and rate of growth are almost fixed. The size of the tooth can be changed by destroying parts of it or by restorative processes of various kinds. Caries activity can destroy all or part of a tooth. The tooth can be returned to its original status or to a different status by many kinds of restorative work. A peg lateral may be enlarged by a jacket crown. The number of teeth present may be changed by extraction or by addition of artificial teeth. At present we seem to be able very little either to inhibit or to accelerate the rate of growth. We see then that while we may produce many modifications upon the dentition through surgical techniques of one kind or another, there is little we can do to produce modification of the individual tooth *so long as it is left intact.* Thus most orthodontic change must be about the teeth and not within them. Other areas in the dentofacial complex likewise have narrow limits within which modification may be instituted without resorting to surgical practice. Little can be done to change the size, shape, and configuration of the mandibular condylar and coronoid processes, the rami, the angles, or the body. Almost equally resistant to change is the complex of structure that constitutes the maxillary apical base. So far as we know we can do little either to stimulate or to retard its growth.

The two parts which offer the widest latitude for modification are the maxillary and mandibular alveolar areas. Genetic phenomena appear to be responsible for the amount of alveolar bone to be provided and the rate, through growth, at which it will be provided. Whether we can stimulate a greater amount to be grown than this remains to be demonstrated. We think, probably not!

We pose then the proposition that effective orthodontic work of all kinds may be done within the limits set by the amount of alveolar bone. An appraisal of the amount available at a given time can be made by direct examination and a conservatively accurate estimate of the amount yet to be obtained though growth can be ascertained by recourse to hereditary evidence.

From the standpoint of treatment hereditary evidence serves to outline further the technique to be used. Some of these treatments may be termed comprehensive and others compromise in accordance with usage already established in orthodontics. In some of the compromise cases, especially those involving extraction of teeth or resection of the mandibular rami, knowledge of heredity is very useful. Fewer departures from standard practice are found, perhaps, in the comprehensive types of treatment. Here information concerning heredity functions primarily to guide expectation of what growth will accomplish.

Finally this is a preliminary report on our findings to date (April, 1942). None of the problems initially posed have been solved in the detail we believe essential for the profession. We know, at least, that hereditary factors are present and important in the dentofacial complex. We have found that knowledge of heredity is useful in guiding the operator in orthodontic practice.

Subsequent work will present case reports in which information concerning heredity is used as a basis for treatment and will present more complete and more precise information on several details brought out in the body of the paper.

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THE SIGNIFICANCE OF "CIRCULATORY CANALS" IN ROENTGENOGRAMS OF THE ALVEOLAR PROCESS

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INTRODUCTION AND LITERARY SURVEY

RENEWED interest in the circulatory canals of the mandible and maxilla has developed in recent years because of improved technique in roentgenography. Although much of the anatomic description of the blood supply of the teeth and jaws dates back to Zuckerkandl's work in 1891,[12] it was not until 1923 that Hirschfeld first described the roentgenographic appearance of "interdental canals."[8, 9] Since then, there have been a number of reports on these structures. In most cases they have been described as normal anatomic features and no consideration has been given to the fact that they can be seen in roentgenograms of relatively few individuals. Because of this occasional appearance, the question of their diagnostic importance has been raised. Can any significance be attached to their presence?

In order to evaluate roentgenograms properly, it is necessary to have a thorough understanding of the anatomy and histology of the tissues under consideration. In dentistry, this implies a comprehension of the nerve and blood supply of the teeth and their surrounding structures, as well as knowledge of their microscopic aspects.

Zuckerkandl described the mandibular artery from the mandibular foramen through the mandibular canal, giving off "interalveolar arteries" and emerging at the mental foramen as the mental artery. Just before issuing from the mental foramen, the mandibular artery gives off an incisive branch which passes forward to supply the region anterior to the mental foramen. In a later publication, Zuckerkandl[13] gave a description of the blood supply of the maxilla, particularly in reference to the maxillary sinus and alveolar ridge.

In conjunction with a study of skulls, Hirschfeld reported the presence of canals in roentgenograms of both the mandible and maxilla. Later[9] he gave an amplified description of those found in the mandible. He stated ". . . radiolucent streaks are found one on each side of the mandible between the central and lateral incisors, where they start variously at some point near the apical half of the roots. The upper end of the radiolucent line is usually quite distinct and may be seen to terminate occasionally in a circular shadow caused by the canal's foramen, which is situated on the lingual plate. Starting invariably with a well-formed foramen in the lingual plate of the aveolar process at some point between the apical half of the central and lateral incisor roots, they dip down obliquely through the cortex into the cancellous part of the mandible.

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Dissection of a wet specimen disclosed the fact that the canals and foramina transmit an anastomosing vessel between the inferior dental and sublingual arteries. The linear and circular radiolucent areas are produced by the interdental canals and foramina which are normal anatomical features and not the result of pathologic bone destruction."

Hirschfeld furthermore stated that several additional canals may be found between the other mandibular incisors. His publications in this field, while mentioning and showing canals in the maxilla, were essentially written to show that vessels and nerves from the mandibular canal pass through these canals and foramina in the lingual alveolar plate of the mandible to anastomose with the sublingual vessels and nerves. No mention is made of their significance when seen in the maxilla or in edentulous areas except in the anterior portion of the mandible.

Diamond[3] stated, "Recent investigations by Dr. I. Hirschfeld have disclosed the presence of nutrient canals running vertically within the alveolar process and communicating with the main channels. Hirschfeld canals are found particularly in the region of the septa of the lower anteriors and occasionally extend as far as the premolars."

Pollia, in 1930[10] described vascular channels as being found in cases of "disuse atrophy" and stated, "These channels are linear or branched sections of radiolucence which appear like miniature inferior dental canals. They generally have more distinct boundaries than the canal. They are most commonly seen in the antrum radiographs and sometimes in areas of disuse atrophy. A very positive sign of disuse in an edentulous area is the presence of one or more vascular channels in the radiograph of the alveolar process, although their occurrence is of no value in the antrum image."

Thompson[11] mentioned: "Nutrient canals and vascular areas of the dental arches appear as fine chain-like radiolucent areas between the shadow images of the roots of the teeth. The blood channels appear as heavy radiolucent lines."

Ennis and Batson[1, 5, 6] described these canals as normal structures illustrating their course in relation to the larger vessels supplying both the mandible and maxilla. In discussing nutrient canals of the maxilla, they state: "They occur in the outer wall of the sinus in a groove or rather large, bony canal, which we notice in the roentgenogram as a radiolucent line running anterior posteriorly through the sinus. In some cases, this line extends forward to its terminal branches in the nasal fossa near the ala of the nose. The canals will be observed as radiolucent lines in the walls of the sinus. They are sometimes in direct communication with the apices of the teeth."

In speaking of these structures in the mandible, they continue: "A thorough study of the internal anatomy of the mandible discloses more clearly the ramifications of the mandibular artery and its numberless branches traversing the cancellated structure of the mandible. Batson in 1933 described the manifold courses followed by the branches of the mandibular artery, the mental artery leaving the mandible at the mental foramen, the incisive branch running forward in the mandible to the symphysis, giving off branches passing upward and making their exits through the nutrient foramina on the lingual side of the mandible posterior to the incisor teeth. Another branch passes

through the lingual foramen on the lingual side of the mandible in the region of the genial tubercle and anastomoses with the lingual artery. The mandibular canal through which the artery passes varies as to its relative position in the structure. In some cases, it may be observed traversing a line near the lower border of the mandible; in other cases, it appears near the apical regions of the teeth; in some instances it is seen superimposed over the apical thirds of the roots of the teeth. It appears on the roentgenogram as a radiolucent area, just as will appear the smaller branches of the mandibular canal through which pass the lesser branches of the mandibular artery. They may be noticed in all parts of the mandible, more numerously in the anterior region. Although for years these smaller canals have been ignored by many dentists, the roentgenologist has brought out their important relation to the entire structural composition of the mandible, and they must be reckoned in considering many phases of operative dentistry. Hirschfeld called them 'interdental canals,' but, as they are not only interdental, but paradental as well; Batson prefers to designate them as "nutrient canals," a term more adequately denoting their functions, for they carry nutrition to the parts by means of the interalveolar arteries. As teeth are lost and resorption of the alveolar process occurs, the nutrient foramen of the mandible approaches the crest of the ridge. Resorption may be so extensive that the outer wall of the nutrient canals may even be lost."

In 1936, Dunning and Davenport[4] described interdental canals as follows: "A term given by Hirschfeld to canals or channels in the mandible, between the central and lateral incisors, one on each side. From their well-defined foramina, in the lingual plate of the alveolar process, they run downward vertically into the body of the bone and transmit an anastomosing vessel between the sublingual and the inferior dental arteries."

In attempting to utilize these reports as a basis for diagnosis, difficulties were encountered. Except for Pollia, all had believed that the appearance of these controls in roentgenograms were normal, although only occasionally seen. Casual professional experience seemed to indicate that they were to be found only in certain types of cases. As no available report presented a reason for this limited appearance, it was deemed advisable to survey some material and attempt a correlation of these roentgenographic signs and clinical conditions.

STUDY OF 1050 DENTURE ROENTGENOGRAMS FOR CIRCULATORY CANALS

A. *Description of Material.*—The material for this study was obtained from the clinical departments of the College of Dentistry and the Dento-Medical Research Department at the George Williams Hooper Foundation for Medical Research of the University of California. A total of 1050 entire denture roentgenograms were collected which were divided into four groups. The first consisted of 800 random cases (Group I), the second of 210 patients who were under treatment in the orthodontic division (Group II), the third included 16 edentulous cases from the denture division (Group III), and, finally, the fourth group consisted of 24 dry mandibles (Group IV).*

*The writer is grealy indebted to Dr. George Hughes of the Division of Denture Prosthesis, who placed these mandibles at his disposal for examination and to Dr. Gordon Fitzgerald of the Division of Dental Roentgenography, for his courtesy in making the dental roentgenograms.

The available material was divided into these four groups in order to obtain information on the frequency of canals in a large random group, and, in addition, to determine if a variation in frequency occurred in an adolescent group with dentofacial anomalies (especially those with lack of occlusion), and if their occurrence in edentulous cases could be related to variations in anatomic form. Partially edentulous areas were omitted from this study because the myriad combinations made it impossible to evaluate them competently.

In those cases where canals were found, further study was made of the oral clinical condition of the patient. In addition, photographs and plaster models were available in the majority of cases to aid in determining the relation of function and the anatomic form of the alveolar process.

The ages varied considerably, ranging from 9 to 68 years for the entire group. In Group I, the range was from 15 to 68, in Group II, from 9 to 17, and in Group III from 29 to 60 years. No ages were available for the dry skulls of Group IV. There were 416 females and 384 males in Group I, 117 females and 93 males in Group II, and 7 females and 9 males in Group III, with a wide distribution of nationalities.

B. *Results.*—A summary of the frequency of the roentgenographic appearance of circulatory canals in Groups I to IV is given in Table I.

TABLE I

FREQUENCY OF CIRCULATORY CANALS IN 1050 ENTIRE DENTURE ROENTGENOGRAMS (GROUPS I TO IV)

LOCATION OF CIRCULATORY CANALS	GROUP I 800 CASES		GROUP II 210 CASES		GROUP III 16 CASES		GROUP IV 24 CASES		TOTAL 1050 CASES	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
Mandible Anterior Area	86	10.75	37	17.6	9	56.25	12	50.0	144	13.71
Mandible Posterior Area	2	0.25	–	–	–	–	–	–	2	0.19
Maxilla	12	1.5	2	0.9	8	50.0	–	–	22	2.09
Antrum	34	4.37	–	–	–	–	–	–	34	3.23

Group I. In the first series of 800 entire denture roentgenograms, the circulatory canals were observed in practically all areas of the alveolar process: the anterior as well as posterior areas of the mandible, the maxilla, and also in the wall of the antrum.

Table I shows that the most frequent observations were made in the anterior region of the mandible with a total of 86 cases, or 10.75 per cent. Only in two instances were canals found in the alveolar process of the posterior area of the mandible. Twelve cases were found distributed in various parts of the maxilla, with no specific preference. Because of the relatively high frequency of the appearance of these canals in the wall of the antrum, they were classified separately and constitute 34 cases, or 4.37 per cent, the next highest to those found in the anterior area of the mandible. In these 34 cases, there seemed to be no uniform type of vault shape nor even a definite relation of the apices of the teeth to the floor of the antrum. In some instances, the course of the vessels could be traced across the entire antrum and in others only a short section could be seen. There seemed to be no connection between these vessels and the teeth

in many cases. The great variation in the position of maxillary teeth in their relation to the antrum, some being in direct contact and others being more than a centimeter removed, undoubtedly influences the appearance of canals in roentgenograms of this area.*

Group II. In this series of roentgenograms of orthodontic patients, 37 cases, or 17.6 per cent, showed these canals in the anterior area of the mandible (Table I). It will be noticed that this is a somewhat higher percentage than that of the older and larger Group I. In only two of the entire denture sets of films were canals noted in the maxilla, and none were found in the posterior region of the mandible. Because many of these cases were from children with incomplete jaw development, it was frequently not possible to demonstrate the antrum.

Group III. This series was composed of 16 patients who were completely edentulous and of whom entire denture sets of roentgenograms were available. In these, 9 showed canals in the mandible and 8 in the maxilla; 5 patients had no canals; 5 in one jaw only and 6 in both jaws. Although this series is small in number, the very high frequency of occurrence of these canals, 56.25 per cent in the mandible and 50 per cent in the maxilla, would seem to be of considerable significance.

Group IV. To amplify the material on edentulous jaws further, 24 edentulous dry mandibles and their roentgenograms were studied. In 12, or 50 per cent, of these, canals were present, which follows the frequency found in Group III.

C. *Comment.*—The results of the above survey of 1050 roentgenograms clearly indicates that circulatory canals are found in all areas of the alveolar process. However, it must be noted that they are far less frequently seen in the maxilla, antrum wall, and posterior region than in the anterior region of the mandible.

When seen in the alveolar portion of the maxilla, the canals usually were in a vertical direction, extending from the interdental bone area upward about a centimeter, and then disappearing. There did not seem to be foramina at the ends, but rather a gradual change of direction leading to a fading out of the shadow in the roentgenogram. In those cases where they were connected with the apices of teeth, they were shorter than when seen between the roots.

In the posterior portion of the mandible below the roots of the molars, a large canal is usually noted which transmits the mandibular nerve and vessels. In some cases, canals can be seen extending from this to the apices of the molar teeth. In two cases, other canals were seen which were between the roots of the teeth in the interdental bone. They did not seem to be connected with the teeth or the mandibular canal and their very rare occurrence in this area seems to point to a mere anatomic variation in which an accessory vessel occurs in the cortical plate of the mandible in the posterior region. There is no apparent signficance to this.

*There is a great variation of methods of making roentgenograms of the maxillary molar teeth and different techniques give different views of the antrum. Most of the cases examined in this series were made with the film approximately parallel to the long axis of the teeth and with the principal direction of the roentgen rays at right angle to this. This reduces the area of the antrum seen on the film, but presents it in a truer relation to the teeth.

Because of the comparatively high frequency of the canals in the anterior area of the mandible, a detailed analysis was made as to possible reasons that might have led to their roentgenographic manifestation. It appeared that there were three of these:

1. Anatomic form and structure of the alveolar process.
2. Anomalies of tooth and bone formation.
3. Lack of occlusal function.

Clinical experience has indicated that in all instances where circulatory canals appear roentgenographically, they can be grouped under these three headings. Representative cases have been selected from each group to illustrate their characteristics.

Fig. 1.

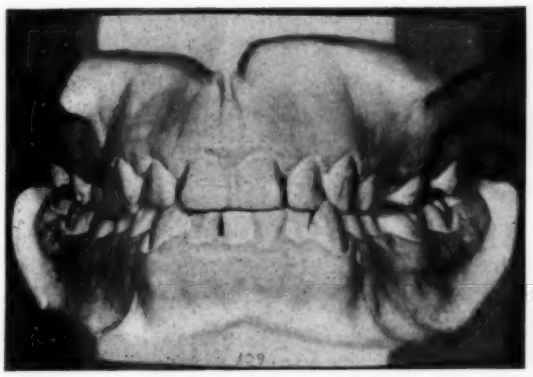


Fig. 2.

Fig. 1.—Depression of labial surface of mandible. Note outline of roots of anterior teeth.
Fig. 2.—Roentgenogram of mandibular incisors of Fig. 1 with canals in interdental bone.

Fig. 3.

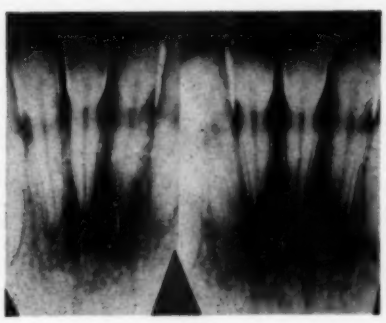


Fig. 4.

Figs. 3 and 4.—Depression of labial surface of mandible and roentgenogram of this area with canals in interdental bone.

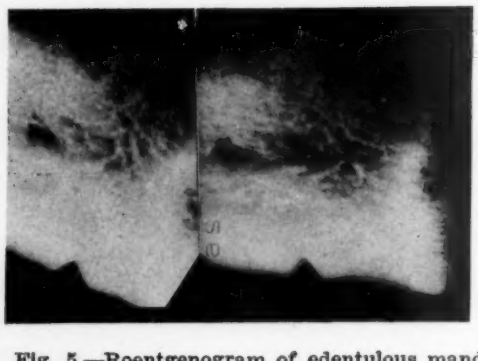
1. Anatomic form and structure of the alveolar process.—Most frequently observed was the presence of a marked depression of the alveolar ridge in the areas where canals were noted. Typical cases are shown in Figs. 1 and 3. Their respective roentgenograms are demonstrated in Figs. 2 and 4.

Mr. P., aged 20 (Figs. 1 and 2) had been treated orthodontically, but in general, when examined for this study, had no serious dental complaint. The roentgenograms showed large canals in the anterior area of the mandible, and study of the plaster reproductions of the teeth and alveolar ridges showed a marked depression of both the labial and lingual surfaces of the mandible in

this area. It was possible to see clearly the outlines of the roots of the mandibular anterior teeth, which indicates an entremely thin labial alveolar plate.

Miss V., aged 30 (Figs. 3 and 4) also had very definite canals in the roentgenograms of the mandible. She had presented for paradentic treatment at the clinic, and during the course of her examination it was found that she was suffering from glandular dysfunctions which probably were responsible in part for her dental complaints. She had never been treated orthodontically. The maxillary anterior teeth had apical abscesses and were extracted before models were obtained, but clinical examination of the mandibular incisor teeth showed that there had been considerable incisal function. The depression of the labial surface of the alveolar ridge can be noted on the cast (Fig. 3).

Fig. 5.



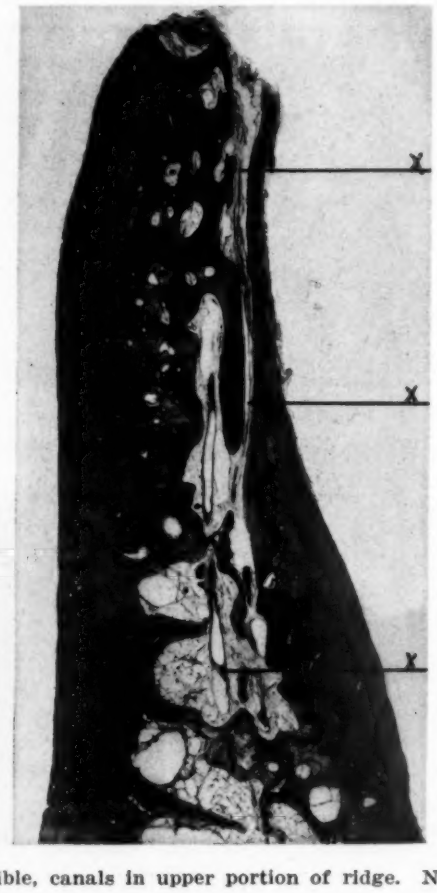


Fig. 6.

Fig. 5.—Roentgenogram of edentulous mandible, canals in upper portion of ridge. Notch on lower border for orientation.

Fig. 6.—Cross section of area over notch, showing course of vessel (*X*) from central marrow cavity through cortical bone.

It was not possible to determine exactly how many of the Group I cases had a definite depression of the alveolar ridge; however, in 39 cases out of 55 in which models were available, this condition was noted, which is approximately 70 per cent. In Group II, 22 out of 37 cases, or 54 per cent, showing canals in this area had this anatomic condition. On the basis of this finding, it seems likely that when the cortical plate is constricted tightly around the teeth, nutrient vessels will be in a position to appear in roentgenograms made of such areas.

In regard to this aspect, Grimm[7] in the Division of Dental Medicine, University of California, who has recently undertaken a comparative histologic and roentgenographic study of the distribution of bone trabeculae in mandibles, made an interesting observation. He found in the roentgenograms of the edentulous mandible a large number of these circulatory canals. By marking the bone structure (Fig. 5), he was able to orientate histologic sections subsequently made from this area.

A section through such a circulatory canal is shown in Fig. 6. It will be noted that the labial and lingual cortical plates have joined together in about the upper third of the jaw, resulting in a very thin compact ridge, practically devoid of marrow spaces. It is interesting to observe that the vessel becomes visible in the roentgenogram only after it has left the central marrow cavity and has penetrated the dense bone layer of the alveolar ridge.

In relating this observation of Grimm to the cases examined, it was found that in many of these with large definite canals there was an anatomically thin ridge. This was due, in some cases, to a depression on the labial or lingual sides or to an underdevelopment of the jaws. The structure of the bone seen in the roentgenograms of these areas was frequently not typical in the arrangement of trabeculae; it appeared washed out, thinner, or as if made up of cortical bone only. The impression is obtained that the cancellous portion of the mandible is diminished in these cases and that the blood vessels and nerves pass in bony canals through the cortical plate.

The cause of this constriction or narrow shape of the ridge may be manifold. Developmental disturbances, atrophic changes, as well as some systemic factors, may influence the anatomic form of the mandible. Following the loss of teeth, with resultant reorganization of the ridge, it is easy to visualize the consolidation of the labial and lingual cortical plates and trapping of vessels and nerves in this dense bone. Clinically, such edentulous cases present thin "knife edge" ridges.

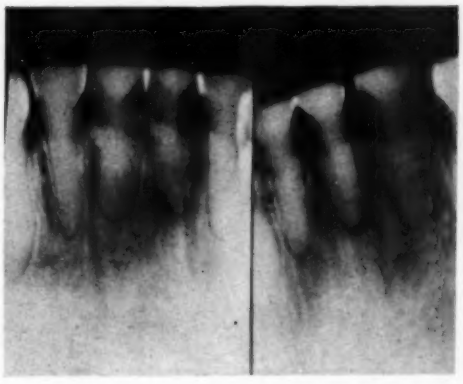


Fig. 7.—Enlarged circulatory canals in a patient with hereditary opalescent dentine.

2. Anomalies of tooth and bone formation.—In this series were cases with unusual conditions in which canals of this type were noted repeatedly.

Mr. B., aged 40, was a typical case of hereditary opalescent dentine (enamel agenesis). The teeth were small, and although generally erupting into normal occlusion, they quickly wore down to the level of the alveolar ridge.

The markedly enlarged canals in this case can be seen in the roentgenogram (Fig. 7). Since this anomaly is comparatively rare, it is difficult to determine whether the canals are pathognomonic or whether they occurred on some other basis.

Miss G., aged 14 (Figs. 8, 9) was seen because of a congenital lack of tooth development (partial anodontia). When examined, only a few deciduous teeth were found. The definite wear on the teeth can be noted in the cast and roentgenogram. This patient was suffering from an endocrinopathy, with ovarian and pituitary dysfunction. When seen after one year during which time the mandibular anterior teeth were removed, the canals were found to be still present and in practically the same place (Fig. 10).

Fig. 8.

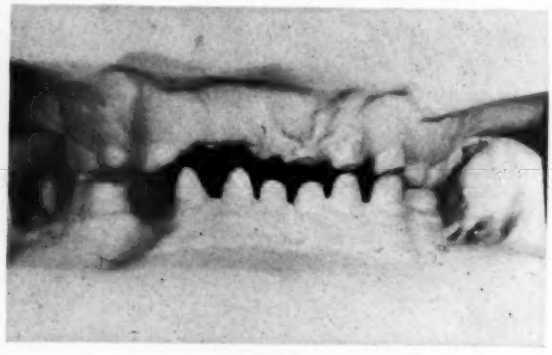


Fig. 9.

Figs. 8 and 9.—Models and roentgenograms of Partial Anodontia, with enlarged circulatory canals.

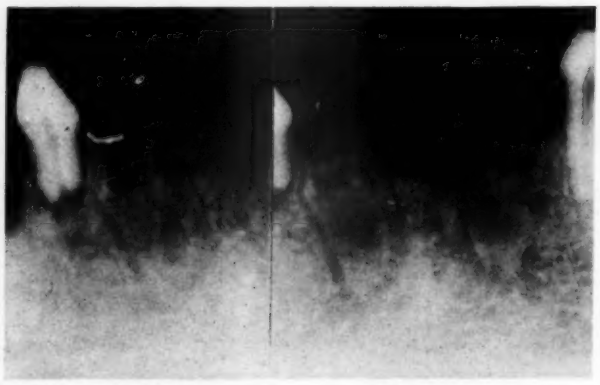


Fig. 10.—Same case as Figs. 8 and 9, canals still present one year after extraction of incisors.

Another case was a patient, Mrs. K., aged 47, with marble bone disease, who presented for general dental care (Fig. 11). There was considerable wear on the mandibular incisor teeth, with several canals present.

Several acromegalic patients were also included in this series. This disease, developing from dysfunction of the anterior lobe of the pituitary gland, shows definite changes in the bones, particularly the mandible. Roentgenograms of the anterior area of the mandibles of two patients are shown: Mr. B., aged 43, had symptoms of acromegaly and in the routine dental examination marked enlargement of the canals in the mandible were noted (Fig. 12). Miss B., aged

28, was also an acromegalic, with enlarged canals in the mandibular anterior region (Fig. 13).

In spite of the fact that these two cases of acromegaly showed a clinical enlargement of the mandible accompanied by osteoporotic changes, it must be realized that the canals may not be directly related to the pituitary dysfunction. The possibility of progressive disuse of the anterior teeth must be given consideration.

Other cases in which there was evidence of canal formation were some that had been described by Becks[2] in his discussion of the problem of root resorption. These were patients in whom systemic disorders were found in conjunction with severe dental and bone changes, and in most instances exhibited marked root resorption. Not all of Beck's cases showed canals, but in some instances they were seen in extreme form. The following two cases are typical of these (Figs. 14, 15, 16).

Fig. 11.—Extensive circulatory canal formation in marble bone disease.

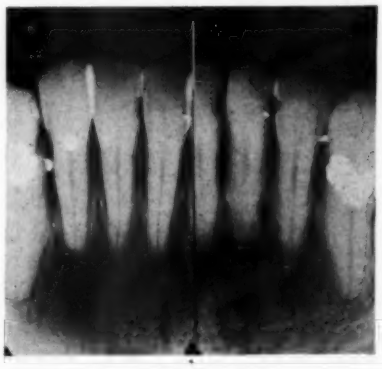


Fig. 12.—Acromegaly, with circulatory canals.

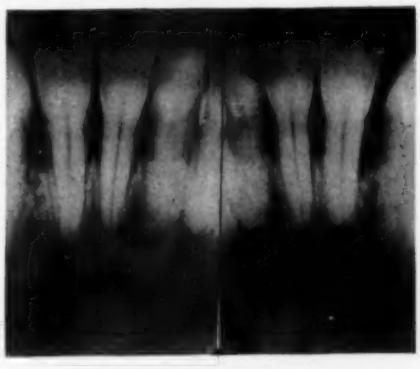


Fig. 13.—Acromegaly, with circulatory canals.

Miss M., aged 36, had been under orthodontic therapy for more than fourteen years (Figs. 14 and 15). Physical examination showed hypothyroidism. The extent of the root resorption and the enlarged canals can be seen in the roentgenograms. The casts showed a definite cupping in of the labial and lingual surfaces of the mandible in the anterior region.

Mr. L., aged 24, who had also received orthodontic treatment (Fig. 16) had one of the most severe cases of root resorption seen during the investigation. He was suffering from a mixed glandular involvement. Clinically, he

had a lack of anterior occlusion and roentgenograms show extensive root resorption and canal formation in the anterior area of the mandible.

The fact that they were not seen in all cases of typical bone disturbances makes it difficult to relate their presence to this factor definitely; however, there seems to be a higher frequency. It is probable that conditions which produce roentgenographically visible changes in the bone structure of the mandible produce related changes in the blood and nerve supply.

Fig. 14.

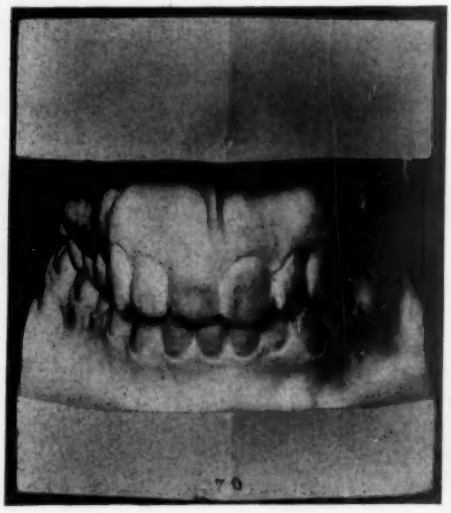


Fig. 15.

Figs. 14 and 15.—Models and roentgenograms of a patient with extensive root resorption and canal formation following orthodontic treatment.

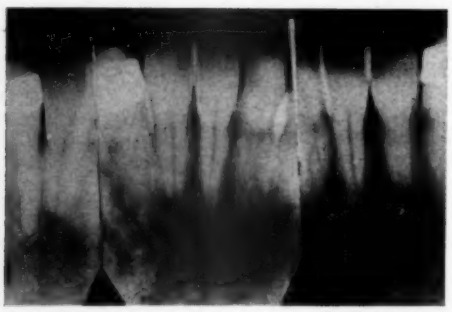


Fig. 16.—Pluriglandular disturbance accompanied by root resorption and extended circulatory canals.

3. Lack of occlusal function.—When this study was begun, it was frequently noted that these structures occurred in a great number of cases where there was no occlusal function. For this reason, special consideration was given to this aspect. Since it was not possible to study adequately the occlusion of all the cases of Group I, efforts were concentrated on Group II, the 210 orthodontic patients for whom casts were available. Of these, 151, or 71.91 per cent, showed definite function in the anterior region, while the other 59, or 28.09 per cent, had marked overjet or other malocclusion which prevented incisal function. Study of the frequency of canals in these two subgroups revealed that 19 of the 151 cases, or 12.57 per cent, had these structures, whereas 15 of the 59, or 25.42 per cent, without function, showed evidence of them.

This would seem to indicate that even though their frequency was twice as great in the latter group, the factor of function may not be of great significance.

SUMMARY AND CONCLUSIONS

A study of 1050 entire denture roentgenograms from a random group of 800 institutional patients, 210 orthodontic applicants, 16 edentulous patients, and 24 dry jaws for the presence of circulatory canals as originally described by Zuckerkandl and later Hirschfeld, showed the following results:

1. They were found in 202 instances, or 19.2 per cent.

2. They were found in all parts of the mandible and maxilla in the following frequencies:

a. Mandible anterior region,	144 cases, or	13.71 per cent
b. Mandible posterior region,	2 cases, or	19.00 per cent
c. Maxilla,	22 cases, or	2.09 per cent
d. Antrum,	34 cases, or	3.23 per cent

3. Three reasons for their roentgenographic manifestation are discussed.

a. Variation in anatomic form and structure were found to be important because these canals were seen frequently in cases with thin ridges, where the marrow cavity seemed to be eliminated. The impression was obtained that they are seen in roentgenograms only when located in solid bone structure.

b. In the presence of anomalies of tooth and bone formations, as well as under pathologic conditions, variations of blood and nerve supply occur and in these cases circulatory canals may be observed.

c. In the series of orthodontic applicants, it was possible to study the factor of function on the anterior teeth. Twenty-eight per cent of the group did not have incisal function. In the cases without function, canals were noted in 25.42 per cent, while in those with function, the frequency was 12.57 per cent.

These studies, therefore, indicate that the presence of circulatory canals in roentgenograms of alveolar bone is an important criterion of anomalies of bone formation and a series of pathologic processes and emphasizes the necessity for detailed study of these factors in each individual case.

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ELECTROLYTIC STAINLESS STEEL POLISHER

MAX J. FUTTERMAN, D.D.S., NEW YORK, N. Y.

THE electrolytic method of cleansing stainless steel is preferred over the mechanical method because it is more convenient, quicker, and effective. Brushes, pumice, rouge, felt wheels, etc., are done away with and a simple procedure is substituted. All that is necessary is to remove any scale that may be present on the appliance, clip the appliance onto the electrode, immerse into the cleansing bath, turn time clock on for about 15 seconds, and rinse in the water wash bath, and the appliance is polished.

Fig. 1.—Electrolytic stainless steel polisher.

In making one-piece lingual appliances, it is often necessary to heat treat areas where sharp bends are made, and then finally to brush flame the entire appliance to anneal it slightly. This carbonizes the metal and gives it a coating which is laborious to remove by mechanical means. In welding various forms of attachments to bands, it is extremely difficult to cleanse the carbonized points at which the welds are made. In soldering of wire to wire where sharp angles

Presented as a clinic at a meeting of the New York Society of Orthodontists, Feb. 24, 1942.

are formed, the polishing is also difficult. These joints are cleansed by the electrolytic process without any difficulty. In soldering operations the scale formed by the flux must first be removed before the action takes place. Bands treated by this method also remain cleaner in the mouth and retain a higher luster. An immersion of the appliance in the electrolytic solution for 15 seconds, will remove all surface impurities and polish the appliance to a much higher finish than can be attained by any mechanical method. This is possible because the electrolytic action in the bath is a shearing one, the current passing from the appliance or band (the positive pole) to the anode at the bottom of the tank.

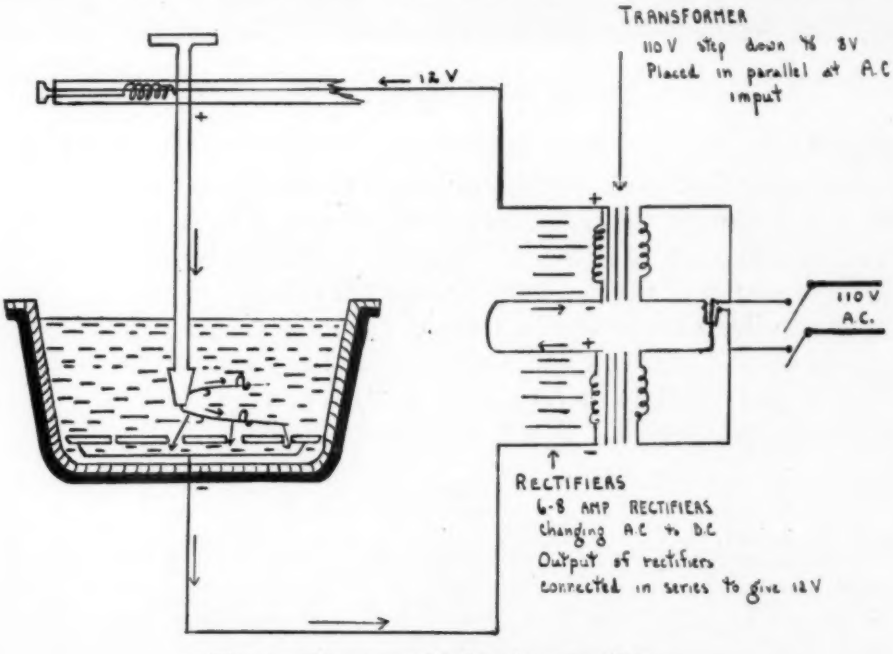

Fig. 2.—Diagrammatic drawing of polisher.

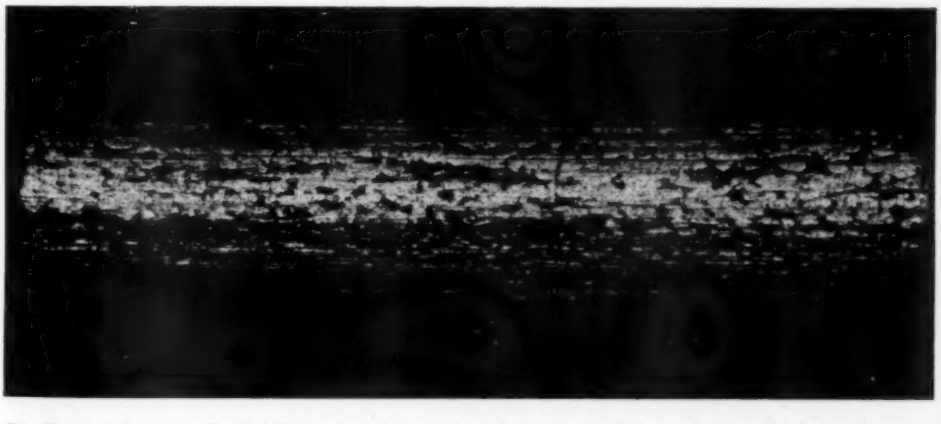

Fig. 3.—Photomicrograph of 0.036 wire before electrolytic polishing. Note pits in surface before polishing. (×100.)

This extremely high finish is desirable not only for its appearance, but the high luster denotes a microscopically smooth surface, which makes for a cleaner appliance. The smoother the surface, the more difficult it is for foreign matter to adhere. The photomicrograph of the electro-treated metal, Fig. 4, proves this point.

This is not a universal cleanser as it does not work on gold. It will not cleanse gold wires, gold bands, or solder. These must still be mechanically hand polished.

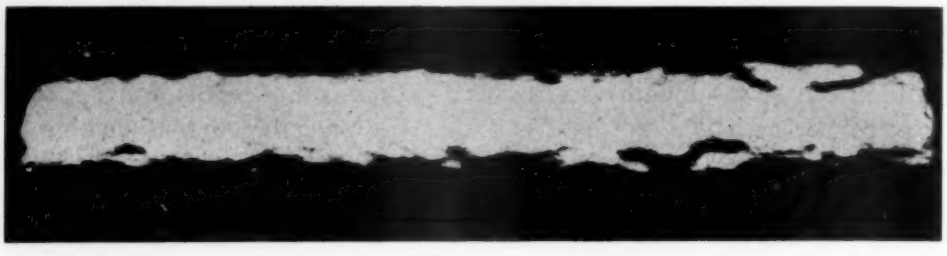

Fig. 4.—Photomicrograph of 0.036 wire after electrolytic polishing. Note extreme smoothness of wire after polishing. (×100.)

Fig. 1 is a photograph of the electrolytic cleanser, while Fig. 2 is a diagrammatic drawing of the cleanser. The customary 110 V A.C. current is used. The current passes through the transformers which steps it down to 12 volts. It is then placed through a 6 to 8 amp. rectifier, further reducing the current to 6 volts. The output of the rectifiers is connected in series to give 12 volts. The current passes down the electrode, through the appliance, through the solution to the negative and then out. The solution used is phosphoric acid (3 parts), glycerine (1 part), and water (1 part). It will be found that if the solution is boiled after original mixing, it will work much better.

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Department of Orthodontic Abstracts and Reviews

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Dental Anomalies and Health: By B. E. Lischer, D.M.D., *The Washington University Dental Journal,* February, 1942.

Under favorable conditions of inheritance, growth, and development the teeth, jaws, and external features of the face frequently unite in such a manner that, taken together, they present a striking symmetry and beauty of form. It has been estimated that at birth the face of an infant is only 42 per cent of its ultimate size. A large amount of the facial growth during childhood and youth takes place in the regions of the mouth and jaws, particularly during those periods when the temporary teeth, and later the permanent teeth, make their appearance. Observation and study of this growth have shown that anomalies, diseases, and loss of teeth influence adversely the development of the jaws and face. In brief, a beautiful, well-formed face requires healthy teeth which are placed in a symmetrical and anatomically correct manner in well-developed jaws, i.e., a denture which functions normally.

Recent studies have shown that anomalies of the teeth and jaws occur in at least 50 per cent of our school children, and that their sequelae are significant for health and normal growth. Clinical observations have shown that premature loss of teeth, prolonged retention of temporary teeth, accidents, habits, and nasal hypertrophies are very potent etiologic factors. We now believe that the prevention and early care of dental anomalies, as well as of oral diseases, have become necessary objectives of adequate dental service.

Dental anomalies seldom involve the entire denture, but when they do, its individual parts are variously affected. It is necessary, therefore, to fix the boundaries of the parts composing a denture and to agree upon their topography, so that we may establish the location and extent of the various malformations.

Imperfect pronunciation in speech is a very common occurrence in patients with dental anomalies. Resonance of the voice, even more than pronunciation, is particularly conditioned by the form of the mouth, the adjacent nasal passages and sinuses, and the symmetrical arrangement of the teeth. Pronunciation in speech may be cultivated, but a clear resonance of the voice can never be acquired if a deformity of oral and nasal structures is present. When dental anomalies are present the lips frequently do not function properly and in time their form and structure may suffer modifications.

Abnormal breathing caused by nasal obstruction and deformity of the denture is also easily recognized. In normal, that is, nasal breathing, the nose cleanses, warms and humidifies the dust-laden air we breathe by means of the

vibrissae and the secretions, which exercise a bactericidal influence on inhaled bacteria. Loss of function of certain facial muscles about the nose and mouth are common and the upper lip, measured in the median plane from subnasion to labrale superius, is frequently too short.

Malposition and malocclusion of the teeth comprise the most common structural lesions of all cases of oral deformity. It is obvious that only a normal mouth can perform normal functions; and by a normal mouth we mean one in which the architectonic form is anatomically correct, as well as one free from disease processes. Malocclusion always implies abnormal function which, almost invariably, leads to other structural lesions.

Again, for the sake of brevity, one can only mention in barest outline, some of the major jaw deformities met with. Transversely, both the maxilla and mandible may present measurable malformations of the dental arches, their alveolar support, and the corpus of each jaw. *Sagitally,* the maxilla and mandible may present deviations, in both anterior and posterior directions, of the dental portions, alveolar structures and the bodies of the jaws. *Vertically,* either or both jaws and their adnexia may be involved. Many combinations of these deviations are possible.

Abnormal development of the temporomandibular articulation and subsequent changes due to mutilation of the denture comprise another considerable group of abnormalities. Dental anomalies are more than mere malocclusion of the teeth because they are the result of pathologic development. They are not processes like disease but relatively permanent conditions. Some of the causes of these conditions are *extrinsic* but many, undoubtedly, are *intrinsic* in nature.

Until very recently the dentist was chiefly concerned with the correction of the more manifest dental malocclusions. To protect the healthy, normal child, that is, to prevent the occurrence of these deformities, is a new concept and presupposes a more comprehensive understanding by the practitioner of the underlying causative processes. Most patients who apply to us for treatment already present disease and anomalies. Inquiry and consultation by healthy individuals is a relatively recent custom and has not achieved any substantial dimensions in dental practice. To provide positive ways and means by which healthy, normal children may avoid dental deformity implies a much better organization and utilization of our knowledge.

A vast majority of individuals who are afflicted with dental anomalies are children who are brought to us by their parents, who seek information and guidance. Unfortunately, such first interviews are frequently allowed to become shopping errands.

The art of procuring an adequate history of a patient requires study and an appreciative regard for its scientific value. The study of orthodontics in the dental school curriculum needs to be more than a training course in the technology of mechanotherapy. Even the correction of dental anomalies as practiced by the orthodontist must be based on a more comprehensive understanding of them as manifestations of abnormal development than has hitherto been deemed necessary. We are confronted by obligations to inquire into the etiology of dental anomalies and to investigate all conditions which cause them.

Dentists, orthodontists, pediatricians, and school teachers should cooperate with parents in procuring the necessary data to prevent these deformities of school children. Certain significant mouth conditions and stages in denture

growth and development need to be assembled into dental norms for all these routine health examinations, so that an objective, quantitative appraisal of each child's dental health may readily be obtained. Obviously, the stages of growth and development of a child's denture, and all deviations which would lead to deformity, should be recorded in such records, so that adequate prophylactic methods may be developed.

J. A. S.

Gum Chewing: Digested from an editorial by M. H. Garvin, *The Journal of the Canadian Dental Association.*

Gum chewing is being popularized over the radio in the most subtle manner imaginable. Recently an American corporation represented its product as being capable of doing away with a gray or dingy condition of the teeth; of ridding one of unpleasant breath; of enabling a person to remain awake and alert while driving his car; of increasing the vitality or strength in men, women, and children due to the dextrose content; of being of material benefit in developing well-arranged teeth in children; of constituting a beauty treatment through the exercise of facial muscles, by keeping the facial muscles young; of aiding in the treatment of a great variety of oral disorders or dental diseases such as gingivitis, alveolar atrophy, acute and chronic Vincent's infection and periodontoclasia. It is interesting to note that this corporation has been forced to enter into a stipulation with the Federal Trade Commission of the U.S.A. in which it agrees to discontinue this type of publicity.

A few months ago another prominent gum company distributed a beautifully illustrated booklet which, it was stated, was prepared for the dental and associated professions. It outlined what was called a functionally efficient means for counteracting pathologic tendencies and stimulating vigorous oral health by chewing gum. A number of our most prominent and most highly regarded dentists were quoted as emphasizing the importance of proper mastication in order to develop healthy jaws; the importance of vigorous chewing habits in order to stimulate healthy tissue reaction, and the importance of function in the treatment of lowered resistance. The inference was that in all of these cases these results could be obtained by acquiring the gum-chewing habit, although probably most, if not all, of these writers had not gum-chewing in mind in any shape or form when making these statements. It was also stated that in a certain orphans' home extensive tests revealed that the systematic chewing of gum tends to prevent the deposit of calculus, and also that bite strength is increased when the masticatory muscles are exercised by the systematic use of chewing gum. It was further stated that in these recent clinical studies it has been shown that tooth scrubbing and the salivary flushing provided by the systematic use of chewing gum definitely help to inhibit the incidence and progress of caries in susceptible children and that is why so many dentists recommend—gum.

As one reads these striking statements about inhibiting the incidence and progress of caries which are of tremendous import to the dental profession as well as to humanity, if such statements are correct, one notices an asterisk referring to the reference column at the end of the booklet where in small type the information is given that these particular statements are taken from an unpublished report from a dental school of a large university. One cannot

help but wonder why such important news should not have been published by this large university and what is the name of this great university and whether other factors are involved which would prove detrimental to the gum manufacturing industry if the report were published.

I am quite aware that certain authors have favored the use of gum to correct certain mouth pathosis. In October, 1911, in *American Medicine,* Kerr points out that chewing gum is very desirable in keeping the buccal cavity clean, particularly in diseases of children. This method was tried for months in cases of typhoid fever, scarlet fever, measles and diphtheria, always with beneficial results according to Kerr.

In the *Dental Register,* December, 1914, Dr. Kleinsoryen of Elberfield states that the treatment of a child where jaws have been neglected in infancy should include a systematic gymnastic for the jaw lasting for a quarter of an hour, at least twice a day. It is stated that a suitable chewing gum may be used for the gymnastic, but that a better medium is a hard carrot which may be chewed to a pulp; or the chewing of grain is also effective and wholesome.

No one could object to the use of gum for a quarter of an hour twice a day under dental direction. In certain special forms gum is advocated for the correction of traumatism. These abrasive gums, however, should always be used under the direction of a dentist. They have also a recognized place in the balancing of artificial dentures.

There are many adults with advanced periodontoclasia who are driving infection into the blood stream every time they attempt to masticate anything. To make this a continuous process during working hours cannot be anything but harmful. At times, following treatment, rest is most beneficial over considerable periods, not excessive exercising.

Dr. Willa Yeretsky, in the *Journal of the Michigan State Dental Society,* cites a case of allergy from gum-chewing. The patient had given up smoking and for the period mentioned had chewed gum constantly. The condition cleared up in ten days following cessation of the gum-chewing habit.

In the November 19, 1941, issue of the *Proceedings of the Mayo Clinic* an interesting case is reported of a 9-year-old boy who was anesthetized with nitrous oxide, oxygen, and ether. A piece of chewing gum measuring 1.5 by 1 cm., which undoubtedly had been eructated from the stomach, was seen lying in the pyriform sinus. This was removed and then a smaller piece measuring 0.5 by 0.3 cm. was seen lying just at the entrance to the glottis. This was also removed. The chewing gum had apparently been swallowed the evening before and had remained in the stomach until the time of the operation. It is quite obvious that in this instance this foreign body could have been easily aspirated into the lung, causing atelectasis or other serious sequelae.

If the saliva has anything to do with the prevention of caries, and most dentists believe that it has, then it is reasonable to suppose that this protective fluid may become weakened and the glands which produce it become less potent as a result of excessive stimulation. I have seen a number of cases where the extent of dental caries could not possibly be accounted for on the basis of diet and which condition seemed to improve definitely after insisting that this habit of chewing gum constantly be stopped.

Then apart from any possible harm which gum-chewing may do to the mouth or to the general health by weakening one of the body's digestive fluids there is the question of appearance, of culture, of character development. There is a certain amount of character depicted in every face and everything possible should be done to develop it along proper lines and not weaken it by gum chewing. I say let dentists awaken to this problem of gum-chewing and instill into the minds of their patients the possible danger of this deplorable habit.

Wax-Resin Compositions for Moulage-Making: By Carl Dame Clarke, Ph.D., *J. Lab. & Clin. Med.* **27**: 1098, May, 1942.

Probably the greatest drawback to the use of wax-resin mixtures is the fact that the resulting cast is broken easily. This disadvantage is overcome when the cast is made of celluloid, rubber, or gelatin mixtures. However, the wax-resin cast is important because it is often used as an intermediate step in making casts of other materials. For example, in a rubber facial prosthesis a wax cast generally is made first and fitted to the patient before the rubber one is made. Necessary changes to obtain perfect fitting are easier to effect in wax than in rubber.

The wax-resin composition used for the positive impression should possess the following qualities:

1. It should be possible to brush or "paint" the positive composition into a plaster or agar mold without having the cast crack or lift from the surface of the mold.
2. The melting point should be so high that it will not be affected by natural heat in the hottest weather. A cast, no matter how thin, should never droop as a candle often does under normal summer temperatures.
3. It should not crack or shrink to any great extent when subjected to the normal cold of winter.
4. It should not crack under sudden changes of temperature, as does a thin layer of hot wax of a high melting point when poured into a cold mold.
5. The shrinkage of the positive wax should be so slight as to cause no cracks when it is shrinking against a rigid mold or after it has been mounted.
6. It should become set, yet remain pliable, at a degree of heat low enough for it to be bent or formed with the hands without breaking or cracking the wax or fear of burning the hands; nor should the composition stick to the hands. This pliability is advantageous, since it often becomes necessary to bend a positive composition into a more desirable shape. For example, it may be necessary to bend the positive to relieve it from some undercut surface on a rigid plaster mold. After the positive has been relieved of such an undercut, it can be rebent to its normal position.

Advantages of Being Able to Apply the Positive Material With a Brush

A positive wax-resin material that can be brushed, spread, or painted, instead of being poured into the mold, offers decided advantages which may be enumerated as follows:

1. It eliminates the danger of the much dreaded border lines, i.e., lines caused by the junction of hot and cold waxes on a smooth surface. The hot and

cold waxes do not blend together, and as a result a line is formed where the waxes of different temperatures meet.

2. The application of the positive with a brush permits a freedom of working without the necessity of lifting heavy molds to turn them in various positions so that the wax will flow evenly and in the same thickness over the entire mold. This same method, when applied to large forms, will prevent a great outlay of physical strength and nerve racking exertion, which also may require the assistance of helpmates to produce the cast.

3. It is far more economical to "paint" a mold than to pour one, because a quantity of wax may be lost or damaged beyond further use by dripping over the edges of the mold while the latter is being rotated, at the same time soiling one's clothing and burning the hands. The composition may fall on the floor, worktable, or any place except where it should go.

4. "Painting in" makes it possible to fill the separate sections of a piece mold before they are put in place and insures the surfaces of being well and evenly covered. After they have been put in their proper places, the positive pieces may be cemented together by the application of more wax, which may be poured or painted into the cracks between the positive pieces.

5. In painting with the positive substance rather than pouring it into the mold, it is possible to apply masses of different materials and colors next to one another in the same layer. By the application of one translucent color over another, interesting and lifelike effects may be obtained. One may spread on the mold a wax composition to resemble the skin in color and texture to the edges of a gapping, moist, reddish wound, which would ordinarily glisten with high lights. This wound area of the cast can be painted in with a different material and color from that of the skin. While this second material may have a wax base, it can be incorporated with other materials, such as various gums, to produce the desired glossy effect.

6. One may prepare a palette from an ordinary muffin pan and fill each cup with positive wax material which has been mixed with a different color. The caster may then use this muffin pan as an artist does his palette in painting a canvas; the caster paints his mold instead. The thin applications of the various colors on this mold must be strengthened or backed up by a thicker layer of wax, then re-enforced with gauze, cheesecloth, or burlap fibers or threads.

In this manner the small blue vessels, which lie beneath the transparent skin, can be incorporated into the cast in the most naturalistic manner. Another indication for the use of color in the same way is a typical hemorrhage under the skin. The fresh flowing blood possesses a different color from stagnant blood. Thus the vessels may be painted red on a thin coating of semi-transparent positive formula which has been applied to the mold. These vessel details are then backed with more composition and finally strengthened with gauze, cheesecloth, or burlap. When the cast is finally removed from the mold, the vessels appear to lie under the skin rather than painted on the surface, as is so often done by the caster. It is interesting to note that red vessels under an ivory colored skin appear blue when viewed through the skin. This may be tested by placing a piece of ivory-colored tracing paper over dark red

marks drawn on white paper. These red marks will seem to be blue, provided the right shade of tracing paper and the proper shade of red are used.

There are unlimited possibilities for the specific use of a positive material that can be painted into the mold rather than one that must be poured. In fact, these advantages are too numerous to mention. However, they will suggest themselves as the worker begins the making of molds and casts.

A simple basic formula for brushing into the mold is as follows:

	PARTS BY WEIGHT
Paraffin (about 55° C.)	8
Carnauba wax	1
Rosin, water-white, lump	8
Red oil-soluble dye	q.s.

The following is a suitable formula for pouring into the mold:

Paraffin (about 55° C.)	8
Carnauba wax (light)	1
Rosin, water-white, lump	2
Red oil-soluble dye	q.s.

*Resin**

In spite of the fact that such work is often called "wax" casting, resin is the basic substance in those formulas which are the most simple to use and have the most versatility. Resin gives the compositions toughness and the quality of bending without breaking; it hardens more slowly, and prevents warping and cracking to a great extent. Common rosin or colophony is generally used for this purpose because of its cheapness. Bleached resin (i.e., water-white) serves better where the chromatic intensity is to be kept low. *The resin content should amount to about half of the mixture,* otherwise brushing of the positive mixture into the mold is sometimes rendered difficult and leaves "lap lines" or border lines. Such lines are formed where a hot mixture meets a cold or set mixture of the positive material and causes a line to appear at the junction of the hot and cold mixtures. Compositions containing a large percentage of resin do not flow easily into fine details unless they are painted into the mold.

If the rosin is powdered or if too much is applied, it may oxidize quite readily, turn dark, and separate from the mixture as a nonsoluble precipitate in the bottom of the pot (see Compounding).

Wax

Low Melting Point.—The viscosity and viscidity, or internal friction, of resins are reduced by the addition of a wax. Such waxes are paraffin, white beeswax, ceresin, and spermaceti. Here again the cheaper product, paraffin, is generally chosen.

High Melting Point.—The melting point of a wax and resin composition may be raised by the addition of a wax having a high melting point. Such waxes are carnauba, montan, and Chinese wax. Carnauba, light, is usually selected. The amount of such a wax that is used in the composition should never be more

*The term "resin" means an amorphous substance that exudes from plants. The term "rosin" indicates the residue remaining after the distillation of oil of turpentine. Generally, common rosin is the resin of the pine tree. It is also called colophony. Gum mastic, gum dammar, and similar substances are also resins but they are not rosins. Gum rosin darkens less rapidly than what is called wood rosin. Both are obtained from the pine tree. Gum mastic or gum dammar could be substituted for rosin (colophony), but these resins are decidedly more expensive.

than one-tenth the weight of the resin; otherwise shrinkage and brittleness of the entire composition will result.

In addition to raising the melting point of a wax-resin mixture, carnauba wax also improves the flowing qualities of the mixture and makes it possible to obtain a sharp impression of skin detail, such as the finger print pattern. If this wax is not included, the resulting cast may be soft and lack sharp, concise detail.

Color

Where it is desirable to have the wax-resin mixture of a basic flesh tone which is composed of red and yellow, a permanent red-oil-soluble dye is added. A yellow dye is not used because the yellow of the carnauba wax and resin is sufficient. The red dye mixes with the yellow of the wax and resin to produce the flesh tint. Of course, such a dye must be used sparingly. A gram or so of dye is dissolved first in about 200 c.c. of turpentine. An eye dropper is then used to place a drop at a time of this solution into the melted wax-resin mixture until the desired shade is reached. From time to time the resulting color may be observed by placing a few drops of the melted mixture on a piece of white paper. When this sets, it is carefully studied in daylight to see whether it is of the basic shade desired. The final monotone wax-resin cast is fixed with shellac or lacquer and then tinted with the proper oil-soluble pigments.

Compounding

Water is placed in the bottom section of a double boiler and brought to a boil. The fire is then cut down until the water just boils. Rapid boiling may cause the mixture to become too dark. This is important, because excess heat or prolonged heating causes the resin of the mixture to darken and separate as an insoluble resin. The waxes only are placed in the top section and allowed to melt completely before the resin is added. At no time must the pot be allowed to boil vigorously while the resin is mixed with the wax. Heating may be continued with occasional stirring, until the resin is melted completely. Prolonged heating causes it to darken. In fact, some workers prefer to melt the waxes thoroughly and then cut off all heat before adding the resin, depending on the stored-up heat in the waxes and the water section of the double boiler to melt the resin. This does not always suffice, but it will serve to impress upon the worker the importance of not getting the resin too hot.

The basic formulas herewith presented may be varied to suit specific purposes or they may be used as specified. Other waxes and resins may be substituted. Those given were chosen because their cost was decidedly less than waxes and resins having similar qualities, such as melting points and viscosity.

Adverse Publicity: Editorial by A. C. Rohde. *J. Wis. State Den. Soc.* **18**: No. 4., July-Aug., 1942.

"Why aren't you treating my daughter according to the new method of treatment advocated in *Time?*" This was the question asked Rohde a short time ago by the mother of a patient. In response to this query, the following editorial was written:

A most fantastic article pertaining to the subject of orthodontics appeared in the March 30, 1942, issue of *Time*. It revealed that yearly 300,000 youngsters

go through the painful and costly ordeal of having crooked teeth clamped into braces to be straightened. It then went on to tell of a hopeful new radical development worked out at Yale Medical School by Dr. Bert George Anderson. It was claimed that by building up a complicated engineering structure of small bands, spurs, and wires in the mouth, it was possible to complete the treatment of some forms of malocclusion in from two days to two weeks.

This mysterious, magnificent method, as elucidated by Dr. Anderson in an article appearing in the A.D.A. magazine for April, 1942, turned out to be nothing more than moving upper anterior teeth, which were slightly lingual to the lowers, into their correct positions. Experienced orthodontists and general practitioners have done this by different methods and in as short a time for the past fifty years. There is nothing new, nothing mysterious about it.

Unfortunately, the article in *Time* gives the reader the impression that something new and advanced has been found at Yale which will be a boon to the children of the country. As a matter of fact, the "discovery" is covered with whiskers.

This adverse publicity is important because the public does not realize that only a very small percentage of cases of malocclusion can be treated by this method. It is important because the public is unaware of the fact that nothing new has been contributed which was not already known. It is important because the public is made to believe that present methods are obsolete. It is important because orthodontists throughout the country are being approached by the parents of their little patients as to why they do not use the methods advocated in *Time*. It is important because publicity such as this is just another "dud" and tends to shake the confidence of the public and undoes a tremendous amount of desirable publicity.

How do these stories rate the "big top"? Is it because essayists and clinicians talk too glibly to reporters in order to get a little ink? Is it because our committees on Public Relations are not consulted by lay magazines or if they are, do they fail to act?

All of us know that the editors of high-class magazines including *Time*, try to enlighten the public by telling the truth. Why is it, then, that in publishing articles of professional nature, they fail to consult with the committees of national societies who would gladly give advice? When editors fail to do this they venture into fields they know nothing about and the article in *Time* is an excellent example. On the other hand, the article with illustrations on the subject of orthodontics which appeared in *Life* about a year ago showed evidence of careful procedure and collaboration.

What can we do to prevent the lay magazines publishing articles containing not only grotesque descriptions of technique but a lot of misinformation as well?

Editorial

Practice Dislocations

In spite of some criticism which makes the point that the medical profession is far short in its war quota, nonetheless both the medical and dental professions are making a fine record in their contributions to the war effort up to the present. Long before the United States entered the war thousands of physicians and dentists were serving on local draft boards throughout the land without pay, examining millions of draftees for physical fitness.

The medical profession particularly is being called upon to supply more man power in proportion to its numbers than is any other occupational group in the country. By the end of the current year one out of every four of America's 160,000 physicians will be in the Armed Forces. Practically every physician able to pass a physical examination satisfactorily and who is under 45 years of age will be in military service. Eventually it is safe to predict that many above 45 years will be included in the Armed Forces.

Many physicians and dentists are unselfishly giving up long-established practices and a satisfactory livelihood to join in the great emergency in which the world finds itself. Not only those who join the Armed Forces but those who remain at home face drastic and important dislocations in their way of life and practice that probably up to now have been little felt as compared with what the future months may hold.

The Federal man power director is now empowered to order the relocation of professional men wherever they may be needed or required in the war emergency; the effect that this will create on private practices when it gets under way will be far reaching.

What of some of the highly specialized departments of practice that up to the present have not received so much attention in the general planning for the civilian emergency? What, for instance, is happening to the continuation of orthodontic practices long established in which the orthodontist has been called to the Armed Forces?

According to reports received the majority of practices so affected have been absorbed by older men in the locality who are not likely to be called into service. It appears that most of these practices have been satisfactorily continued as a result of private negotiation between the man called into service and the man chosen to act as his successor in continuing the practice. Urgency of this question is even now becoming acute in some quarters. In one city where four men have been conducting orthodontic practices as a specialty, one man regarded as satisfactorily equipped by experience and training has been left to carry on. It is reported that in order for him to complete his task it has become necessary for him to work continuously, through Sundays, holidays, and at night part of the time.

On account of the wide variation in technique employed by different men in orthodontic practice, the question of the continuation of a case to successful termination by a successor is a serious one. Obviously, when possible, a successor should be chosen who is entirely familiar with the technique used by the man who is called into service. This qualification should lighten the load in no small degree for the man who is to carry on and complete the cases. It would seem expedient then for those who expect to be called in the future to plan far ahead so that the transition may be made with as little static as possible, and with the added advantage that patients will experience as little hardship as possible.

The AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY has been asked to offer help and suggestions through its columns on this subject that will serve as a constructive influence. The editor knows of no better way than to open the columns of the JOURNAL for reader suggestions and comment during this emergency.

H. C. P.

News and Notes

Research Section, American Association of Orthodontists

At the present time there is no opportunity afforded men interested in orthodontic research to meet together and discuss their problems. At the last meeting of the American Association of Orthodontists it was proposed that the Research Committee be empowered to arrange a portion of the program for the next annual meeting.

The program will consume one-half day and will be restricted to ten- or fifteen-minute reports by men actively engaged in research in orthodontics, or in its allied fields. Time will be allowed for discussion of all presentations, in order that the greatest benefit may accrue to those participating. All institutions and all individuals are invited to request time.

For further information or time reservations on the program address communication to

ALLAN G. BRODIE, Chairman
Research Committee, A. A. O.
30 North Michigan Avenue
Chicago, Illinois

New York Society of Orthodontists

The next or Fall Meeting of the New York Society of Orthodontists will be held at the Waldorf-Astoria Hotel, New York City, Nov. 9 and 10, 1942.

Prize Essay Contest

The Research Committee of the American Association of Orthodontists has been empowered by the Board of Directors to conduct a prize essay contest. The prize has been set at $200.00 and will be offered annually until further notice. The terms of the competition are as follows:

Eligibility.—Any student enrolled in a recognized university, or any person who has completed his or her formal education in orthodontics not more than three years prior to Jan. 1, 1943, is eligible to compete for the prize.

Essay.—The essay must represent a piece of original research having a direct bearing on the field of orthodontics. It may relate either to a biologic or clinical problem and may represent material that has been offered in partial fulfillment of the requirements of a graduate or postgraduate degree, or any graduate, postgraduate or undergraduate contest. No previously published essay will be accepted. All essays must be in the hands of the committee by Jan. 31, 1943. If no essay is deemed worthy by the committee, the prize will be withheld.

Award.—The prize-winning essay will be accorded a place on the scientific program of the annual meeting of the Association, at which time the prize will be awarded. The Association will retain publication rights on the first three choices.

For further information, address

ALLAN G. BRODIE, Chairman
Research Committee, A. A. O.
30 North Michigan Avenue
Chicago, Illinois

WPB Decision on Stainless Steel Materials

Through the efforts of Drs. George M. Anderson, W. Paul Hoffman, and Charles Smith the JOURNAL is able to print the copies of two letters sent to the chairman of the committee of the A. D. A. on materials and supplies that will be of much interest to orthodontists. The subject discussed in the following letters is stainless steel materials for orthodontists.

WAR PRODUCTION BOARD
WASHINGTON, D. C.
Sept. 10, 1942

Room 4090 Railroad
Retirement Building

Dear Dr. Anderson:

This is a correction to our letter sent out this morning in regards to the mechanics necessary to enable the orthodontists to obtain their stainless materials.

It will be necessary, because of the amendment to M-21-d, passed in March, for the dentist to have a higher rating than the A-10 granted under P.100. Therefore, it becomes necessary to change the mechanics as follows:

The Orthodontist will be required to file on Form PD-1-A and will be assigned a preference rating of A-1-J to conform with Order M-21-d. Supply houses and distributors may obtain their inventory stock by applying on form PD-1-X, to which a preference rating of A.1.A will be assigned.

It is expected, however, that soon this order will be further amended, so as to eliminate the restrictions that make it necessary for the doctors to apply for preference ratings.

Thank you for your very kind cooperation in this matter. Please call on us if we can be of any further assistance.

Very truly yours,

HERBERT B. SHOR, Analyst
Dental & Orthopedic Section
Health Supplies Branch
Division of Industry Operations

. . . . . .

Room 4090, Railroad
Retirement Building

Dear Dr. Anderson:

You will be glad to learn that the matter of making stainless materials available for use in Orthodontia has been favorably concluded.

The mechanics for obtaining this material will be as follows: The Supply House or distributor who deals directly with the manufacturer of these materials may apply for priority assistance on form PD 1-A and then sell to the dentist, school, clinic, etc., on a preference rating of A-10 under order P-100. Copies of P-100 are included, in case you are not familiar with this order.

We trust that your committee will take care of disbursing this news and we would also appreciate your including a personal admonishment for the doctors to refrain from purchasing more than their actual requirements, which we will try to make available to them at all times.

Very truly yours,

HERBERT B. SHOR, Analyst
Dental & Orthopedic Section
Health Supplies Branch
Division of Industry Operations

Encl:
HBS:am

Public Relations Bulletin

In keeping with the wide reader interest among the members of the American Association of Orthodontists in the subject of public relations and inasmuch as the Southwestern Society of Orthodontists, as well as other regional societies, has seen fit to continue public relations activities subsequent to the announcement of the inactivity of the public relations committee of the A.A.O., the JOURNAL herewith publishes the most recent bulletin circulated by the Southwestern Society. The Southwestern Society has been very active in doing something about the conflict between school hours and professional service for children, as the following bulletin reveals.

EDITOR

SOUTHWESTERN SOCIETY OF ORTHODONTISTS

Bulletin

TO: ALL MEMBERS

Physicians and dentists and especially orthodontists are again facing the problem of school attendance vs. professional services.

This year, more than ever before, it threatens to have a damaging effect upon safe and efficient practice unless adjustments are made to compensate properly for the increased demand and responsibilities which the civilian practitioners must assume as a sequence of the war effort.

With the orthodontist's practice almost wholly limited to school children, this group feels the restraining effect of school attendance more keenly than any other.

Since adjustments are the order of the day to coordinate civilian and military activities more effectively for the benefit of the public welfare, it is only fitting that the orthodontists in their respective communities take the lead in obtaining a modification of the rules and regulations governing school attendance in relation to medical and dental services.

Present conditions make it an excellent time psychologically to accomplish something constructive in this direction.

A reprint is enclosed for your further information and guidance.

We suggest that this problem be presented to your local dental society and your local medical society with the idea of having them endorse the movement and select a committee, composed of two medical men and one dentist (preferably an orthodontist) to effect negotiations properly with the school authorities.

A committee of five, made up of three medical men and two dentists (one an orthodontist) might be found preferable. This increased number, however, would make a little more cumbersome arrangement.

A few extra copies of the "Progress Report on School Attendance and Professional Services" are available upon request if you want them. A few copies may be found useful as an aid in presenting the problem to others.

A practical and wholly satisfactory solution is mainly dependent upon the rules and regulations which are adopted. They should exert only the minimum amount of interference with routine school work and be acceptable to all concerned. It is furthermore essential that all the principals, teachers, parents, and practitioners be made thoroughly acquainted with the rules and the manner in which they are expected to function. If the practitioners will provide themselves, at their own expense, with mimeographed copies of the publicized official announcement like the enclosed sample and use them as announcements to circularize the parents of their child patients, it will be conducive to a more effective understanding.

The method adopted in Oklahoma City for solution of the problem has met with no criticism and has been found highly satisfactory to all concerned. H. E. Wrinkle, Superintendent of Schools, 400 N. Walnut St., Oklahoma City, Oklahoma, will certify this statement upon request and your Public Relations Committee will be glad to furnish you with any additional information you may desire.

This subject was discussed at the Board of Directors meeting at Dallas on Aug. 23, 1942, and the Board directed that this problem be brought to the attention of the entire membership.

Sincerely yours,

T. W. SORRELS, Chairman
CURTIS WILLIAMS, Secretary
Public Relations and Military Affairs Committee

. . . . . .

Office of

SUPERINTENDENT OF SCHOOLS

Oklahoma City, Oklahoma

Bulletin Number 5 October 3, 1941

CALENDAR

(Following Item Extracted):

Rules Governing School Attendance

Last year a committee was appointed consisting of J. B. Greene, Ethel Liebhart, Charles Evans, and Lee Nuzum to meet with a committee from the County Medical and Dental Societies to consider certain rules governing school attendance. This committee submitted an excellent report. This report has been approved by all concerned and is hereby submitted for your observance as a part of the rules and regulations of the Oklahoma City Public Schools.

A. That all school children needing the services of a doctor should be encouraged to secure the service.

B. That an absence less than one-half day should not be recorded where an appointment for medical treatment has been kept.

C. That absence due to illness or to keeping appointments with doctors should receive no penalty.

D. That written requests from the parent be presented to the school:

1. When the child wishes to leave the building to keep an appointment for medical service.

2. When the child has been to see the doctor before coming to school and wishes to return to class.

E. That attendance awards should be discontinued.

Great Lakes Society of Orthodontists

The annual meeting of the Great Lakes Society of Orthodontists, scheduled for Oct. 26 and 27, 1942, at Toledo, Ohio, has been postponed, as will be all other meetings of this Society for the term of the war.

C. EDWARD MARTINEK,
Secretary-Treasurer

Notes of Interest

Dr. Claude R. Wood, immediate past-president of the A.A.O., is now on duty with the Army Air Forces with the rank of major and is stationed at the Gulfport Air Base at Gulfport, Miss.

Dr. Hammond L. Johnston, former associate and successor of Dr. Harry E. Kelsey, is in Australia with the Johns Hopkins Hospital medical unit. His address is: Capt. H. L. Johnston, 0-407421, General Hospital # 11, A.P.O. # 927, c/o Postmaster, San Francisco, Calif.

The AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY feels that its readers would be glad to be informed of the change of address and location of any orthodontists who have entered the armed services. If this information will be sent direct to the editor, 8022 Forsythe Blvd., St. Louis, Mo., it will be published from time to time in the *News and Notes* pages of the JOURNAL.

OFFICERS OF ORTHODONTIC SOCIETIES*

American Association of Orthodontists

President, J. A. Burrill - - - - - - - - - - - 25 East Washington St., Chicago, Ill.
Secretary-Treasurer, Max E. Ernst - - - 1250 Lowry Medical Arts Bldg., St. Paul, Minn.
Public Relations Bureau Director, Dwight Anderson
- - - - - - - - - - - - - - - - - - 292 Madison Ave., New York, N. Y.

Central Association of Orthodontists

President, Harold J. Noyes - - - - - - - - - 55 E. Washington St., Chicago, Ill.
Secretary-Treasurer, L. B. Higley - - - - - - - - 705 Summit Ave., Iowa City, Iowa

Great Lakes Society of Orthodontists

President, Henry D. Cossitt - - - - - - - - - - 942 Nicholas Bldg., Toledo, Ohio
Secretary-Treasurer, C. Edward Martinek - - - - - - 661 Fisher Bldg., Detroit, Mich.

New York Society of Orthodontists

President, E. Santley Butler - - - - - - - - 55 Locust Ave., New Rochelle, N. Y.
Secretary-Treasurer, Norman L. Hillyer - - - - Professional Bldg., Hempstead, N. Y.

Pacific Coast Society of Orthodontists

President, Ben L. Reese - - - - - - - - - - - Roosevelt Bldg., Los Angeles, Calif.
Secretary-Treasurer, Earl F. Lussier - - - - - 450 Sutter St., San Francisco, Calif.

Rocky Mountain Society of Orthodontists

President, George H. Siersma - - - - - - - - - 1232 Republic Bldg., Denver, Colo.
Secretary-Treasurer, Curtis L. Benight - - - - - - 1001 Republic Bldg., Denver, Colo.

Southern Society of Orthodontists

President, M. Bagley Walker - - - - - - - - 618 Medical Arts Bldg., Norfolk, Va.
Secretary-Treasurer, E. C. Lunsford - - - - - - 2742 Biscayne Blvd., Miami, Fla.

Southwestern Society of Orthodontists

President, E. Forris Woodring - - - - - - - - - Medical Arts Bldg., Tulsa, Okla.
Secretary-Treasurer, R. E. Olson - - - - - - Union Nat'l Bank Bldg., Wichita, Kan.

American Board of Orthodontics

President, William E. Flesher - - - - - 806 Medical Arts Bldg., Oklahoma City, Okla.
Vice-President, Frederic T. Murlless, Jr. - - - - - 43 Farmington Ave., Hartford, Conn.
Secretary, Bernard G. deVries - - - - - - - - Medical Arts Bldg., Minneapolis, Minn.
Treasurer, Oliver W. White - - - - - - - - 213 David Whitney Bldg., Detroit, Mich.
James D. McCoy - - - - - - - - 3839 Wilshire Blvd., Los Angeles, Calif.
Joseph D. Eby - - - - - - - - - - 121 E. 60th St., New York, N. Y.
Claude R. Wood - - - - - - - - - Medical Arts Bldg., Knoxville, Tenn.

Harvard Society of Orthodontists

President, Harold J. Nice - - - - - - - - - 475 Commonwealth Ave., Boston, Mass.
Secretary-Treasurer, Edward I. Silver - - - - - - - - 80 Boylston St., Boston, Mass.

Washington-Baltimore Society of Orthodontists

President, Paul W. Hoffman - - - - - - - - 1835 Eye St., N. W., Washington, D. C.
Secretary-Treasurer, Stephen C. Hopkins - - - - - 1726 Eye St., Washington, D. C.

Foreign Societies†

British Society for the Study of Orthodontics

***President,* S. A. Riddett**
***Secretary,* R. Cutler**
Treasurer, Harold Chapman

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

†The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis, Mo., U. S. A.

DR. J. A. BURRILL, PRESIDENT OF THE
AMERICAN ASSOCIATION OF ORTHODONTISTS.

UNIV. OF MICH.